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Volume 53 Number 5

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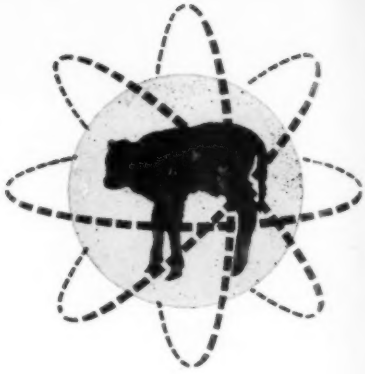

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Reprint from the JOURNAL of the American Veterinary Medical Association, vol. 124, No. 922, Jan., 1954, pp. 19-20.

A Note on the Use of Surital Sodium Anesthesia in Swine

H. W. DUNNE, D.V.M., Ph.D., and Captain S. C. BENBROOK, V.C., U.S. Army
East Lansing, Michigan

THE NEED for a satisfactory anesthetic in swine practice has long been recognized by veterinary surgeons and research workers throughout the nation. Until recently, most of the anesthetics available seemed to be contraindicated in some phase of application. They proved to be either toxic, resulting in frequent deaths, or the duration of anesthesia was too long and incurred time-consuming and struggling association. Floundering and struggling associated with recovery from anesthesia of

½ inch board, in the standing pose on the snout. The animal was directed slightly downward, immobility and fully covered a point between the shoulder and shoulder blades. When the needle entered the appearance of blood in the syringe, was then slowly injected, the solution being given under light pressure. The injection was completed in approximately 1 cc of the solution per pound of body weight. The injection was made rapidly so as to minimize struggling of the animal. The injection was made into the external jugular vein. As soon as the needle was withdrawn from the vein, pressure was applied to the point of injection. Although there were some difficulties encountered in the use of this anesthetic, the results were

Use of Surital Sodium and Curare in Small-Animal Surgery

W. O. Brinker, D.V.M., M.S.
Michigan State College
East Lansing, Mich.

Reprint, The North American Veterinarian, Vol. 32. December, 1951.

Reprinted from VETERINARY MEDICINE, Vol. XLVII, No. 3, March 1952

Surital Sodium And Morphine Anesthe In Canine Surgery

W. C. YOUNG, JR.,* D.V.M. and F. E. EADS D.V.M., M.S., Detroit, Michi

THE use of surital** sodium (sodium 5-allyl-5-(1-methylbutyl)-2-thiobarbiturate), a recently developed ultra-short acting thiobarbiturate, has been reported by several workers. It could be called the thiomalogue of seconal** just as pentothal** sodium could be considered the thiomalogue of nembutal**.

In most instances, intravenously. Howe Dillon and Denson' administration in ve in this manner has t nary medicine.

In five experim

Surital Sodium Anesthesia in Canine Surgery

H. B. ROBERTS, D.V.M.; W. E. WENDT, D.V.M.; C. C. WAGNER, D.V.M., M.Sc.; T. F. REUTNER, D.V.M., M.S.

Reprint from the BULLETIN of the American Veterinary Medical Association, Vol. CXVIII, No. 888, March, 1951, pp. 181-184.

Reprint from the JOURNAL of the American Veterinary Medical Association, vol. 122, No. 912, May, 1953.

Dosage Studies of Surital Sodium in Dogs

ROBERT F. BORGMAN D.V.M., M.S.
Asheville, North Carolina

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TABLE I—Results of Surital So

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Intrathoracic Anesthesia In Cats

MARK STERNFELS, D.V.M., Mount Vernon, New York

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Surital Sodium, A New Anesthetic and Hypnotic
Studies in Dogs

T. F. REUTNER, D.V.M., M.S., and O. M. GRUHZIT, M.S., M.D.
Detroit, Michigan

Reprint from the Journal of the American Veterinary Medical Association, Vol. CXIII, No. 858, October, 1948, pp. 357-360.

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F. E. EADS, D.V.M., M.S., Detroit, Mich.

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covery. They estimate that the approximately 1.5 times that of the dog. Similar results were Kelly, et al.[14] Surital accu slowly in dogs than does pen there appears to be less satura toxification mechanisms whe

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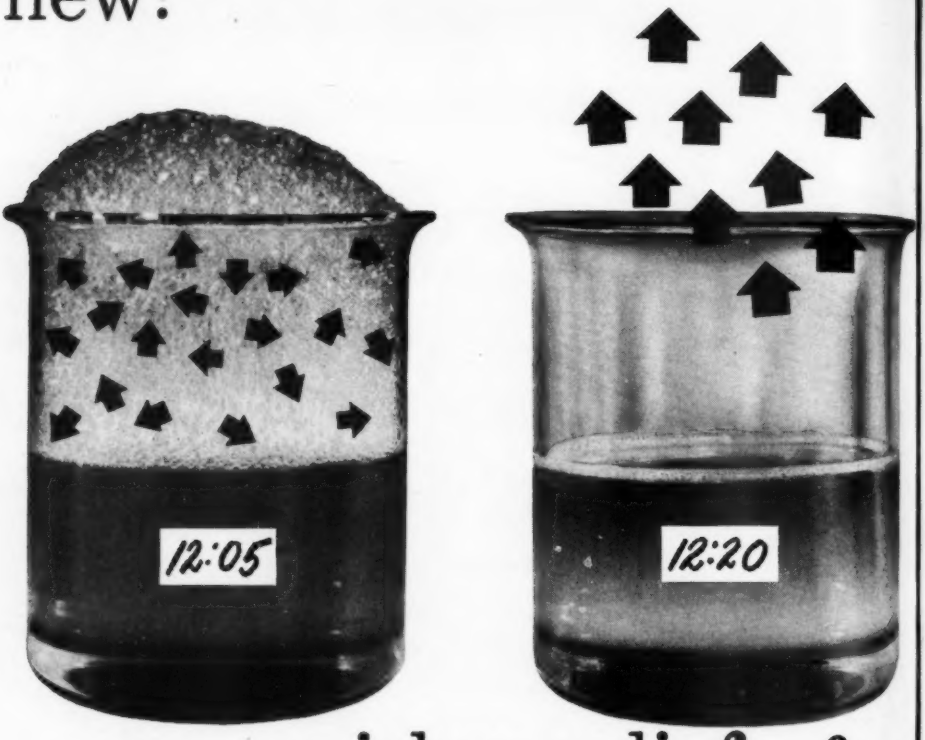

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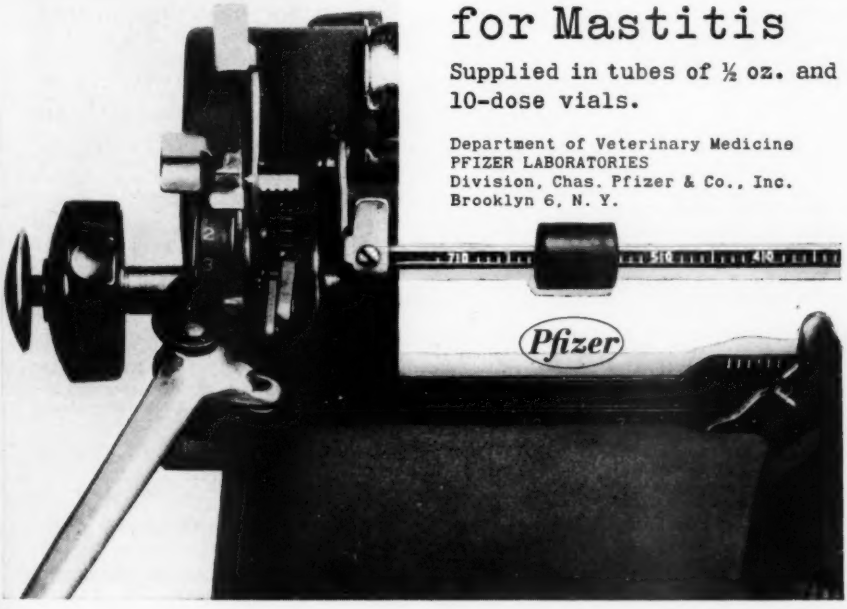

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TRICHLORETHYLENE ANESTHESIA IN THE CAT

Verner L. Johnson, Ph.D., Paul A. Klavano, B.S., D.V.M., Roy Wright (Veterinary Student), and Daniel Sax, A. B.

Improperly employed trichlorethylene anesthesia in the cat may cause serious cardiac damage.

OBSTRUCTION IN THE COLON OF HORSES

E. W. Ellett, B.Sc., D.V.M., E. W. Jones, M.R.C.V.S., Ph.D., and Lester Johnson, A.S., B.S., D.V.M.

The authors outline operative procedures for the relief of true or false fecaliths in horses. Modern surgical technics and shock prophylaxis make surgical intervention in these cases practical.

INTRAMUSCULAR NEMBUTAL IN CANINE ANESTHESIA

Normal Bleicher, B.S.

Herein is emphasized that Nembutal is a safe agent for intramuscular use and that this method of administration has advantages under specific circumstances.

VETERINARIAN-PHARMACIST RELATIONSHIP

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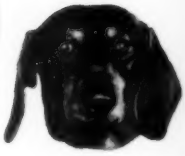

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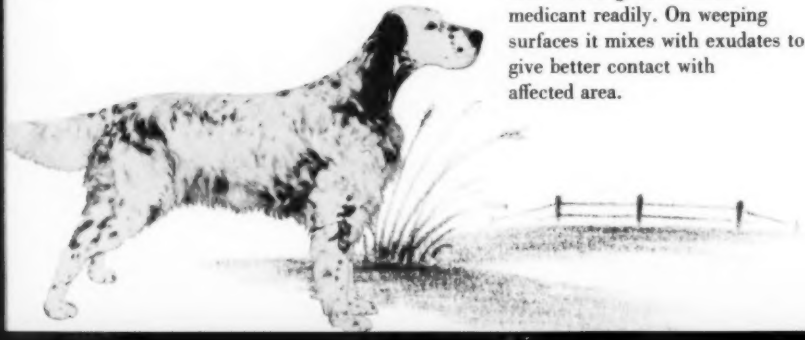

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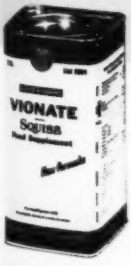

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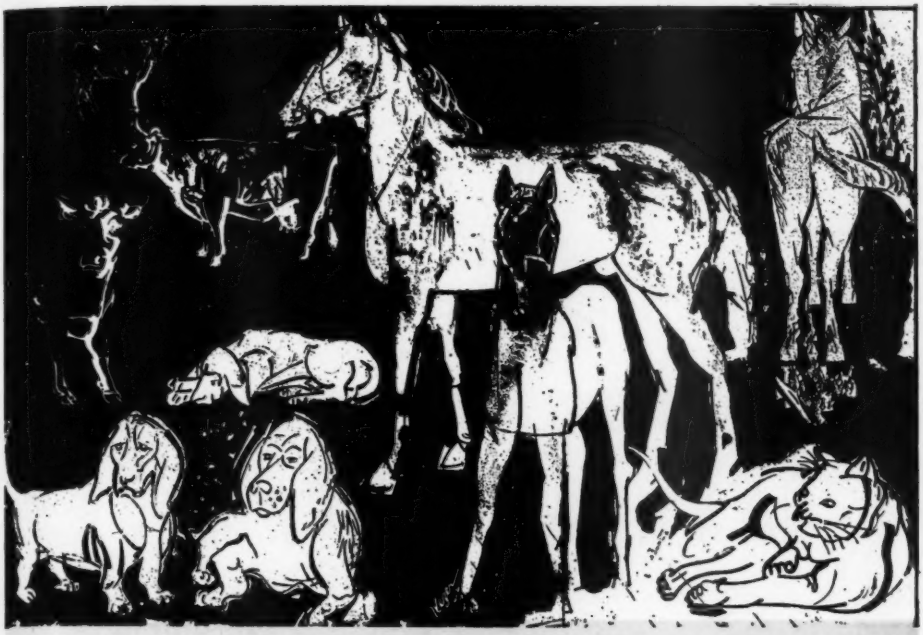

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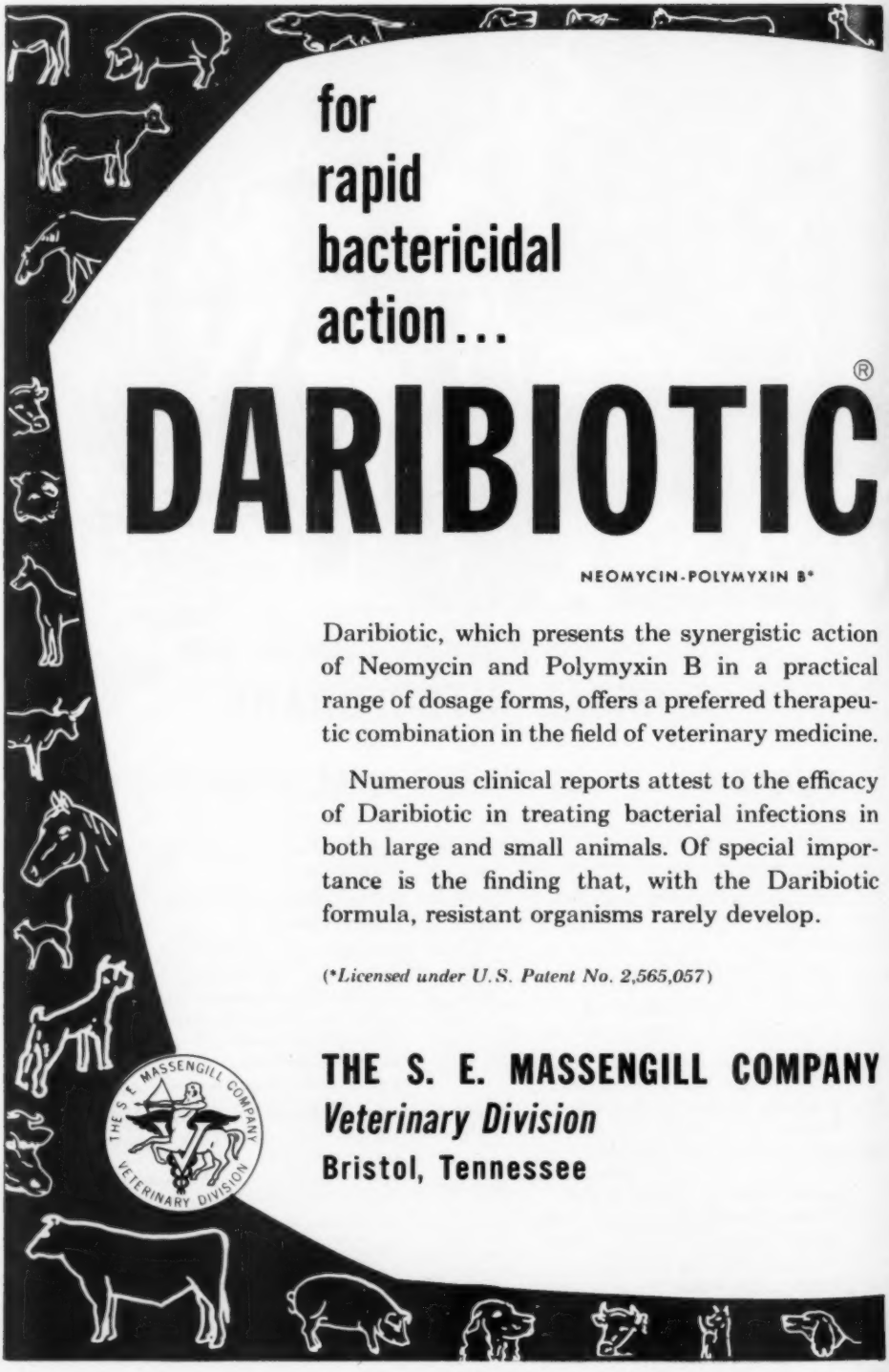

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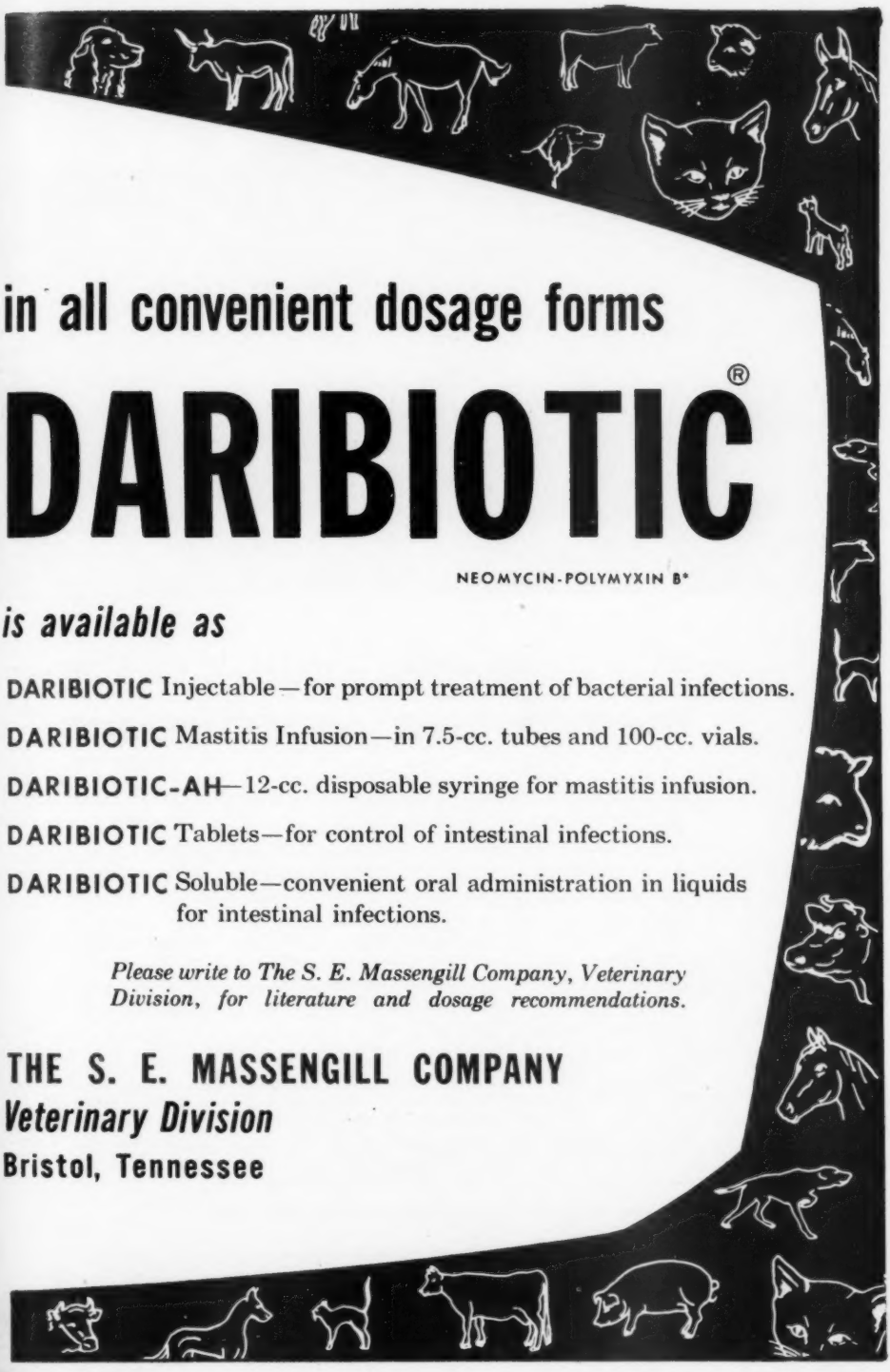

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in chronic respiratory disease of poultry

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VETERINARY

provides bactericidal action at the site of infection

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—highly potent

Prepared from cultures selected for their ability to elaborate highly potent antigens when proper attention is given to their exacting growth requirements.

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Adsorbed, flocculated, and concentrated through action of *Gel 21*, a specially prepared aluminum hydroxide gel, made exclusively for Fort Dodge Laboratories.

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The active immunizing factor is enhanced by adjuvant action of the *Gel 21*, retarding release of antigen into the tissues and prolonging antigenic stimulation.

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Take a long, hard look at this sketch. Then look down the road about ten years. Will today's much-needed, highly respected veterinarian be in this picture of tomorrow—or will he have gone the way of the Indian and the buffalo?

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This situation could greatly reduce the use of your services. And since the future of the livestock industry depends on a strong and thriving veterinary profession, the promotion of products for the control of livestock diseases should be confined exclusively to the veterinary profession.

That's why we hope you'll give a lot of thought to where you buy your supplies. The ethical house sells only to graduate veterinarians; others sell for use by untrained persons. As for Affiliated, we have unalterably cast our lot with that of the Graduate Veterinarian.

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News Comment

Popular Dogs

Judging by American Kennel Club registrations for 1957, the ten most popular breeds of dogs rank in the order named: Beagle, Chihuahua, Dachshund, Boxer, Poodle, German Shepherd, Cocker Spaniel, Collie, Pekingese, and Boston Terrier.

The Rhodesian Ridgeback, so named because of the ridge of hair growing in opposite direction along its spine, is the first new breed admitted to American Kennel Club registration in ten years.

"Punch" of Livestock Rations

University of Minnesota's Dr. R. J. Meade presented a dramatic demonstration of half a century of progress in swine feeding at the University's Farm and Home week.

Litter mates were divided into four pens at weaning (33 lb.) and fed typical rations of 1908, 1930, 1953 and 1957-58. Pigs themselves dramatized the "punch" of today's rations for growth and gain. After 62 days the pig fed 1908 rations weighed 40 lb., the 1957-58 fed pig, 132 lb.

Algae Meal

Sewer-grown algae meal was the subject of a recent investigation by nutritionists. The meal contains 40% crude protein but early trials indicated that residual aluminum was detrimental to growth. Practicality of using this material needs further investigation.

Iowa Bans Feeder Pigs

Iowa State Department of Agriculture has placed a ban on shipment of feeder pigs into the state because of a "dangerous disease situation approaching the emergency stage."

Traffic in feeder pigs has been large during winter months, though normally as many as 600,000 feeders are brought into Iowa for sale annually. The Department seeks more effective laws governing the importation of feeder pigs.

Broiler Production

Statistical data released by the USDA indicates that broiler production (as measured by 22 states that sell 89% of the total) reached a record 1,295,000,000 birds. This is 8% above the number produced in 1956.

Georgia ranked first in total production—a whopping 261,000,000 birds.

Poultry Inspection

Veterinary inspection of poultry for wholesomeness is admittedly the most promising development anticipated by the poultry industry. Correction of some sloppy practices are certain to result in improved acceptability of market products by consumers.

Rations and Mastitis

Illinois workers reported no relation between quantity of protein in the ration and abnormalities of the udder of dairy cows or of the milk. High protein rations did not aggravate existing udder abnormalities, nor did it throw chronic animals into clinical cases.

Beef Imports

Beef and veal imported into the United States for October and November 1957 totaled 53,733,255 pounds, an increase of 33,394,999 (60%) over the same period a year before. During the first 11 months of 1957 imports of beef into the United States totaled 191,852,211 pounds as compared to 100,713,896 pounds for the same period in 1956. This represents an increase of 91,138,315 pounds or about 46%.

Farm Animal Inventory

Cattle inventory January 1, 1958 was the smallest since 1952, according to USDA report. Cattle numbers at 94,000,000 were down 1%, or 535,000 head less than a year earlier. Milk cows declined 2% to slightly less than 22,500,000 head, the smallest since 1928. Beef cow numbers declined 1% whereas all other beef stock were up from a year ago. Hog numbers remained practically unchanged. Sheep and lamb inventories were up 2%; horse and mule numbers declined 6%; chicken and turkey inventories declined 5 and 6% respectively. Total inventory value of all livestock and poultry on farms and ranches—up 27% from a year earlier.

Red Meat Storage Holdings

A seasonal gain of 26,000,000 pounds in storage of red meats was reported during January, bringing the total to 429,000,000 pounds. Net gain last year was 11,000,000 pounds; the average gain during the month is 59,000,000.

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STREPTOKINASE—HUMAN PLASMINOGEN—STREPTODORNASE

Dog with chronic, generalized, bacterial dermatitis. Nose lesion was 4 years old—treated with ointments, antibiotics, lotions and cautery without effect.

Same dog one month later following 5 days' treatment with VARIZYME and POLYOTIC Tetracycline intravenously followed by soothing ointments topically.

VARIZYME is a stable, vacuum-dried powder containing two streptococcal enzymes and plasminogen of human placental origin.

By enzymatic action VARIZYME liquefies fibrin, clotted blood and viscous exudates. Use VARIZYME to reverse inflammatory process and remove inflammatory barrier.

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VARIZYME is indicated for abscesses, slow-to-heal wounds, edema, ulcers, cellulitis and hematoma and other inflammatory conditions. It is used in the treatment of pneumonia, chronic cough, otitis, metritis, mastitis, dermatitis, foot rot, actinomycosis and sinusitis.

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Quantity	Package
12 or 36	12,500 Units Vials
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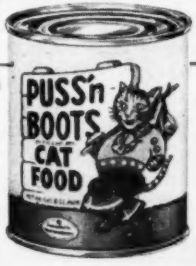

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- controls the intractable animal without physical force
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animal	oral	intravenous	intramuscular
DOG	4 mg./10 lb. b. i. d.	5 mg./20 lb.	5 mg./20 lb.
SWINE		10 mg./100 lb.	10 mg./100 lb.
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NOTE: Refer to Schering literature for complete information regarding dosage, side effects, precautions and contraindications.

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SWIVINE with serum provides protection that is immediate, continuous, lasting—*and economical.*

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IN LARGE OR SMALL HERDS.....

*SWIVINE**

immunizes rapidly, solidly

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The test outlined gives promise of a simple procedure for testing immunity levels to specific diseases. In this respect, the technic described may become a useful diagnostic procedure for practitioners.
In this preliminary report, the authors describe the procedure and their experience with the gel precipitin test using known virus cultures and antisera of determined titers.

The Gel Precipitin Test

I. Its Technic and Applications in Veterinary Medicine

E. J. LAZEAR, Ph.D., A. H. KILLINGER, Ph.D.
M. B. HAYS, M.S., and H. ENGELBRECHT, D.V.M.

Product Development and Diagnostic Laboratory
Fort Dodge Laboratories
Fort Dodge, Iowa

THE gel precipitin test is a new laboratory tool for the demonstration of the combining power of an antigen with its specific antibody. In this test, the antigen and antiserum are allowed to diffuse toward each other through a layer of agar. When the diffusing antigen and antiserum meet in equivalent proportions, an obvious precipitate forms.

The test was originally employed in studies on bacterial toxins and antitoxins by Oudin[1] and Ouchterlony.[2] More recently, it has been adapted to the study of viruses. Diseases such as foot-and-mouth disease,[3,4] hog cholera,[5,6] myxomatosis,[8] and vesicular stomatitis[6] have been investigated. Bacterial toxins, soluble antigens and viruses have been studied in our laboratory.

Test Procedure

The diffusion medium contained 1.75% Noble's special agar (Difco), 0.85% NaCl and 0.01% merthiolate. Essentially this is the medium recommended by Wilson and Pringle[7] except that the concentration of agar has been increased by 0.25%. In our hands, this concentration did not interfere with or block any specific reactions and had the advantage in physical manipulation. Approximately 25 to 30 ml. of molten agar is poured into a 9 cm. petri dish and allowed to solidify on a level surface.

Cup Size and Placement

Cups are cut in the solidified agar by means of a stainless steel tube, 0.7 cm. in diameter, sharpened on one end to form a cutting edge. Agar plugs are removed by the application of a vacuum to the end of the tube opposite the cutting surface.

Another cutting tool consists of six tubes permanently mounted around a center tube. Each radial tube is 0.5 cm. from the central tube and spaced equidistant.

When this tool is pushed into the agar, seven cups are cut with one operation. This

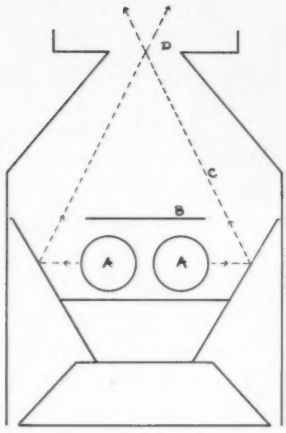


Fig. 1. The laboratory equipment used in the preparation and observation of the gel precipitin tests and a cross-section drawing (right) of the viewing light. Note the application of the darkfield illumination principle in that direct rays of fluorescent tubes (A) are blocked by the baffle (B), allowing only reflected light (C) to pass through the viewing slit (D).

tool is merely a template since the agar plugs usually remain in the cups and must be removed in a second operation. The single tube with vacuum is used to remove the plugs. (See fig. 1.)

Care must be taken when using the cutting tools so that the sides of the cups will be vertical. The vertical plane of the precipitate will parallel the vertical side of the cup. Any distortion in the wall of the cup will be reflected in the precipitate.

Once the plugs are removed, the bottom of each cup is sealed with a drop of molten agar. This step is quite important since the antigen and antiserum must diffuse through the agar and not seep out under it.

The optimum distance between cups is variable and must be determined for each system studied. For screening procedures 0.5 cm. is used. Since the diffusion rate of molecular elements of a substance through the agar is dependent upon their concentration, it is logical that serum having a high titer will travel farther from the cup than will serum of low titer. If one has potent sera the distance between cups can be increased. If sera are of low titer, distance between the cups must be reduced.

By using 25 to 30 ml. of agar per plate and making the cups 0.7 cm. in diameter, 0.1-0.2 ml. of material can be deposited in each cup without overflowing. It is important that material be contained within the small cut-out cup and not be allowed to either seep out under or overflow across the top of the agar.

Incubation

After antigen and antiserum have been placed in the cups, plates are incubated at 37 C. over a pan of water to prevent dehydration. It is not essential to use incubator temperature. Satisfactory results have been obtained when plates were incubated at room temperature. However, rate of diffusion is partially dependent upon temperature, and increases as temperature increases. Most systems gave positive reactions after 16 to 24 hours at 37 C. These same systems required 2 to 3 days at room temperature before comparable reactions were observed. The fact that accurate results were obtained more rapidly at 37 C. should not be interpreted to mean that all systems work better at 37 C. It is entirely possible that room or refrigerator temperature might be optimum for other systems. Regardless of temperature selected, plates should be maintained at constant temperature for the duration of the experiment. This is especially important if one is to compare results of duplicate experiments performed at different times.

Reading Test Results

A good light source is imperative. Reflected light directed up through the plate at an oblique angle to the line of sight is best. For preliminary screening, one may use a fluorescent bulb with shade

For best photographs, the lamp and housing illustrated in figure 1 were developed.

This apparatus was developed along the principle of the darkfield condenser. Direct rays of light from fluorescent tubes are blocked by a center shield. The only light that is emitted through the slit at the top has been reflected at least once inside the housing. Best results are obtained when the center of the petri dish is at the focus of the reflected light rays. The camera is suspended directly over the slit, perpendicular to the surface of the plate.

Dyes

Although it is possible to see lines of precipitation in the agar without them, dyes incorporated in the agar are helpful, especially if photographs are to be taken.

Wilson and Pringle[7] recommended 0.06% methyl orange and Mansi[6] 0.03% of the same dye. Either concentration proved satisfactory for visual observation; both leave something to be desired for photography.

Carbol fuchsin, diazine green, congo red, and methylene blue have been employed in concentration up to 0.06%. For visual observations, congo red is preferred at a concentration of 0.012%. For photographs on black and white film, 0.03% methylene blue is preferable.

Recording

Photographs are preferred for permanent records of reaction. However, there is some doubt about reproduction until the film is developed and printed. The use of a polaroid camera might eliminate this disadvantage. Another disadvantage in photographs is that some faint lines of precipitation are insufficiently resolved to be recorded on the negative.

For recording results in laboratory notebooks, a plastic stencil shaped like the multiple cutting tool has been designed. The cup size of 0.7 cm. was maintained but the distance between the cups was increased to 1 cm. Any test can be reproduced in a laboratory note book and the precipitate drawn to scale.

The plastic stencil is preferred to mimeographed or printed record sheets because of its adaptability. However, if one type of test is used extensively, printed forms would be advantageous.

Applications of the Test

There are a number of reports in the literature dealing with the relative merits of different strains of *Erysipelothrix rhusiopathiae* as immunizing agents. It is generally agreed that virulence is not necessarily related to antigenicity and that those strains which produce soluble antigens are best for bacterin production. In addition, it has been reported that products absorbed on aluminum hydroxide are superior to products without the compound.

Antigens selected for test were formalized broth cultures of *E. rhusiopathiae* with and without added aluminum hydroxide gel. In addition, one modified live-culture product was tested and one chemically inactivated adsorbed with calcium phosphate, tri-basic.

Antiserum was the standard product supplied by the United States Department of Agriculture.

In this study, the agar plate was set up with four radial cups spaced equidistant and 0.5 cm. from the central cup. For reference, the radial cups were numbered clockwise from 1 to 4, the uppermost cup being number 1. Standard undiluted AIQD antiserum was placed in the center cup, (C). Formalized bacterin was placed in cup 1, bacterin with aluminum hydroxide gel in 2, the modified live culture in 3, and the calcium phosphate adsorbed product in number 4.

Object of experiment was to test: (1) Concentration of soluble antigen in the different products, and (2) adsorptive capacity of aluminum hydroxide as compared with calcium phosphate. This, also, shows that the gel precipitin test can be used in experiments with two or more variables.

After 24 hours at 37 C., lines of precipitation were visible between the central cup and cups 1 and 4. After 48 hours, a line between C and cup 4 had fused with the line between C and cup 1. Also, a precipitate was visible now between C and cup 2. After 96 hours, lines of precipita-

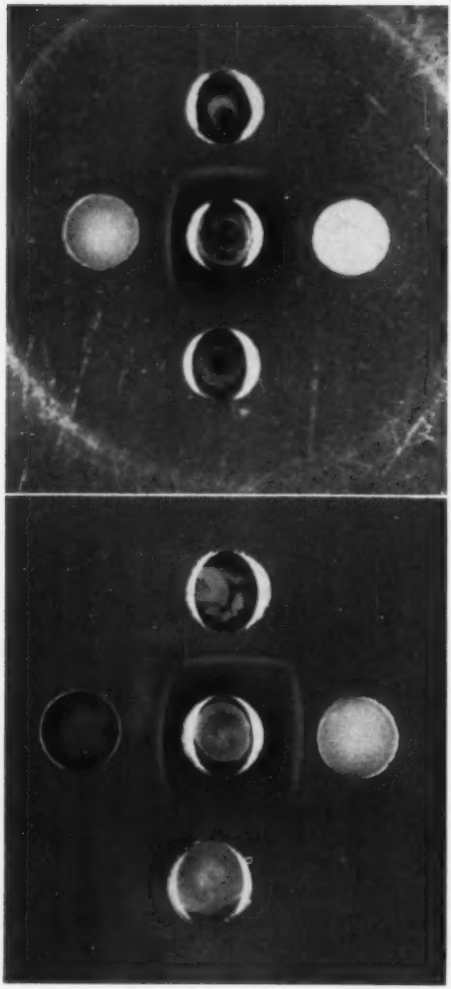


Fig. 2. Four *Erysipelothrix rhusiopathiae* products tested with the standard antiserum: After 24 hours incubation (upper); after 72 hours incubation (lower).

tion between C and cup 4, C and cup 1, and C and cup 2 have fused at their adjacent ends to form a three-sided box around the central cup. Figure 2 illustrates the test plate after 24 hours incubation, and again after 76 hours.

The early appearance of the line of precipitation between C-4 and C-1 indicates that these products have sufficient soluble antigen to produce precipitation with the antiserum at the zone of equivalence. The fact that the C-4 line appeared closer to the antiserum than did the C-1 lines suggest that the former product had more soluble antigen than the latter. The C-4 line changed little after 24 hours while the C-1 became longer and heavier with increased incubation. Maximum concentration at the zone of equivalence was attained later for this product. The delayed appearance of the C-2 line demonstrates that the soluble antigen is absorbed firmly to the aluminum hydroxide and released slowly. The early appearance of the C-4 line suggests that the soluble antigen is loosely adsorbed to the calcium phosphate.

The appearance of a precipitate between the antiserum and the modified live culture product was variable. In most experiments, no visible precipitate appeared. When the precipitate did appear, it was not visible until after 72 hours incubation and even then the line was faint.

The fact that the C-4, C-1, and C-2 lines fused at their adjacent ends indicates that all of these reactions were due to the same antigen. The C-3 line was faint and was not long enough to reach the C-4 or the C-2 line so homogeneity of this antigen is not known. For a detailed description of diffusion rates and homogeneity of antigens, readers are referred to Oudin's report, Methods in Medical Research, vol. 5.

The template cutting tool is useful when one wishes to test a number of sera or serum dilutions against the same antigen or a number of antigens against one serum. By placing the antigen in the center cup, it is possible to test six sera or six dilutions of one serum simultaneously, or six antigens may be placed around one serum sample.

Without using dilutions of serum one can evaluate relative strength of different sera by position of precipitates. Precipitates are closer to the antigen cup in trials with sera having a high concentration of antibodies than one of lesser concentration. In the latter, precipitate will be closer to the antiserum cup. Conversely, it is possible to evaluate virus growth in tissue culture. Poor growth results in no visible precipitation or

line close to the antigen cup. As the virus concentration increases in tissue culture, precipitate line is formed farther and farther from the antigen cup.

In the plate pictured in figure 3, six different infectious canine hepatitis antisera are tested against infectious canine hepatitis virus. Each of these sera give a satisfactory test when tested by the serum neutralization test in tissue culture. The virus was infected tissue culture fluid collected after five days growth in dog kidney tissue culture. At this time, there was extensive cytopathology of the tissue culture cells.

Position of the precipitates show that the sera are of nearly equal titer with the exception that the serum in the Number 3 cup is a little closer to the center cup (antigen) indicating relative high titer or antibody concentration.

Three positive and three negative sera (fig. 4) are shown in a test with another tissue culture antigen. The position of the precipitate reveals that the antigen contains less virus than the one used for the other test, since precipitate formed closer to the antigen cup.

Another application of the template cutting tool is in the testing for homogeneity of antigens. To demonstrate this point, toxins of *Clostridium perfringens* were used.

Whole broth culture filtrates of *Cl. perfringens* Types C and D were used as antigens. These filtrates were placed in alternate cups around the central cup which contained standard type D antitoxin. (See fig. 5 and 6)

Multiple lines of precipitation between the toxin and antitoxins may be noted. One of the lines is coalesced at its ends to form a hexagon ring around the center cup. This coalesced ring shows that these two toxins have at least one antigen in common. Lines of precipitate which cross and do not coalesce are caused by antigens which are not shared by these toxins.

Presence of multiple lines of precipitation with *Cl. perfringens* toxins is not surprising, since each type of organism produces more than one toxin. Also, mouse protection tests with the type D antitoxin indicate that this antitoxin would protect against the type C toxin.

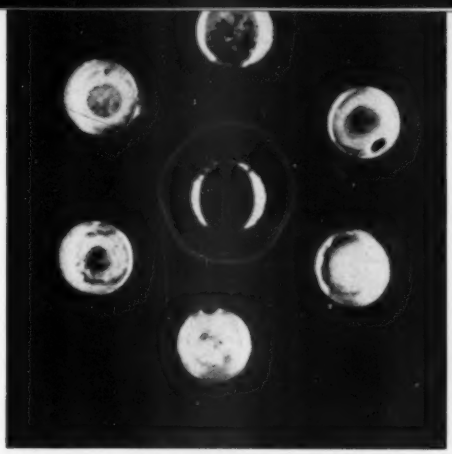


Fig. 3. Anti-infectious canine hepatitis antisera tested with infectious canine hepatitis virus, tissue culture origin. All six sera exhibit positive reaction.

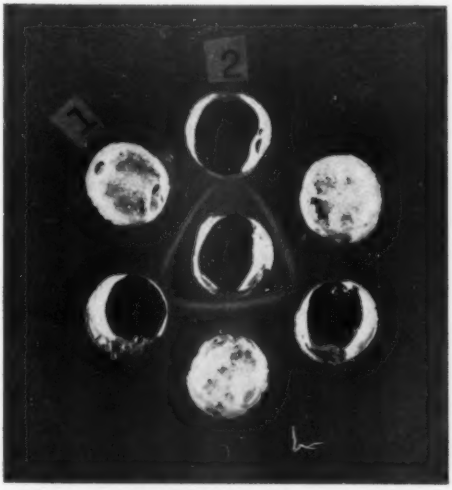
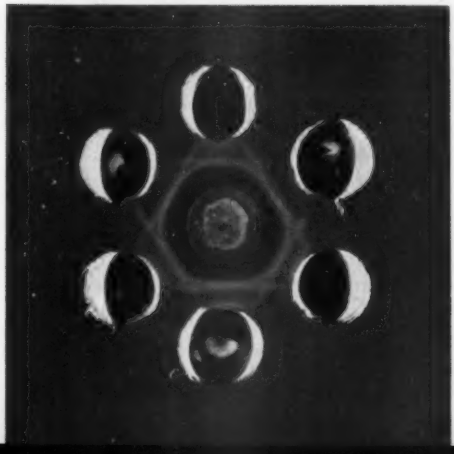


Fig. 4. Illustrating three positive reactions (infectious canine hepatitis antisera) with three normal sera against infectious canine hepatitis virus, tissue culture origin.

Fig. 5. Plate showing the cross reactivity of *Cl. perfringens*, type D antitoxin with *Cl. perfringens*, type C and D toxins.



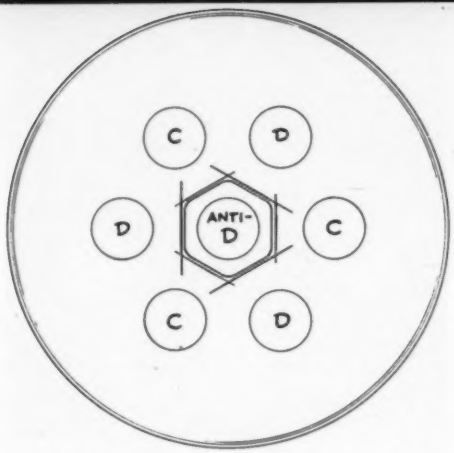


Fig. 6. Laboratory drawing interpretation of fig. 5.

Discussion

This report describes the technic of the gel precipitin test as it applies to veterinary medicine. The test is based on the fact that a visible precipitate forms at the zone of equivalence formed between diffusing antigen and antiserum. Therefore, the test is only as good as ability to observe the precipitate once it has formed. For this purpose, a light has been devised using the darkfield illumination principle, making possible observations under constant light.

As another aid for observation and recording, different dyes have been investigated in various concentrations. For visual observation, congo red in a concentration of .012% is preferred. However, for photographic work, .03% methylene blue gave best results.

For routine laboratory use, the test has embodied advantages of simplicity of operation, adaptability, and versatility. In addition, information of a quantitative and qualitative nature is obtained.

As an example of the adaptability of the test, six sera or two dilutions of three sera or three dilutions of two sera may be tested with one antigen by placing the antisera in the six cups surrounding the central antigen cup. Conversely, six antigens can be tested with one standard serum. The positions of the lines of precipitation will give an indication of the relative titer of antiserum or antigens. Also, qualitative information is obtained from precipitates. If lines of precipitation coalesce into a ring around the central cup, all reactions are caused by one antigen combining with its specific antibody. If lines cross, reactions are due to more than one antigen-antibody system. Number of lines of precipitate indicate the minimum number of antigens and antibodies which are combining.

Another example of the adaptability of the test is that it is possible to test four dilutions of serum against three different antigens on one plate. For this test, the agar plate is set up with three rows of cups with three cups per row or nine cups in the form of a square. Each row of cups is 0.5 cm. apart and each column is 0.5 cm. apart. One antigen is placed in the center cup with antiserum placed in the four cups which are 0.5 cm. from the center cup. Another antigen is placed in the corner cups diagonally across the square. The third antigen is placed in the remaining cups on the opposite corners of the square.

If the antiserum reacts with all three antigens, positive reactions will appear as three sided boxes around the antiserum cups.

In addition to the data presented, some preliminary studies have been made on a number of other diseases and this material will be presented when the studies are completed. Preliminary studies with hog cholera have been encouraging and discouraging.[5] Commercial hog cholera antiserum has been tested with success but substances other than antibodies diffuse into the agar and obscure specific reactions. Also, hog cholera antiserum contains other antibodies.

Pancreatic tissue was selected as the virus source for the hog cholera test. Spleen and blood, although rich in virus, contain excessive hemoglobin which diffuses into the agar and obscures specific reactions. The pancreas is not perfect as a virus source since it, also, contains diffusible substances.

Specific reactions with hog cholera form quite rapidly, (usually 12-18 hours) and fade rapidly (within 48 hours). Most of the other systems studied do not exhibit maximum reactivity until 48 hours and do not fade until after four days.

Results, therefore, have been encouraging because we have been able to demonstrate reactions between hog cholera antiserum and virus from animals diagnosed as having hog cholera. On the other hand, results have been discouraging because of positive reactions which are probably not due to hog cholera virus and because of the technical problems of non-specific diffusing substances.

Summary

The technic and applications of the gel precipitin test for the detection of specific antigen-antibody reactions is described. Agar cutting tools, recommended dye concentrations and a suitable light source are described for optimum readability and photography of specific reactions.

Adaptability of the test has been demonstrated using examples of *E. rhusiopathiae*, infectious canine hepatitis, and *Cl. perfringens*, Type C and D. Information on the relative titer of a number of antisera, the amount of soluble antigen in different products, the efficiency of adjuvants, the concentration of virus in tissue culture, the cross reactivity of antitoxin and the homogeneity of antigens has been obtained by this technic.

The potential use of this test procedure in studies on other diseases, namely hog cholera, is discussed.

Addendum

Subsequent reports outlining specific application of the gel precipitin test as a diagnostic procedure will appear in subsequent issues of VETERINARY MEDICINE as technics are perfected. These reports await thorough trials and observations on specific sera and antigens having known titers.

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Tympany Due to Foam Formation in the Rumen

DURING 1957, a series of articles was published in a leading German veterinary journal[1] by Prof. E. Lienert and Dr. G. Kienel of the Veterinary College of Vienna who, for the first time, succeeded in duplicating conditions which permit the study of tympany and its treatment in the laboratory. The investigators concentrated on the problem of (1) gas (foam) formation, particularly forthy bloat due to fermentation processes and (2) gas expulsion which, in the case of bloat, becomes a physical-chemical foam problem.

Frothy bloat occurs chiefly in animals pastured on legume fields. Legumes contain a number of foam formers — saponins, proteins, and mucilaginous substances. The experiments were made with a foam tester consisting of a 600 cc. cylinder with thermostat and circulatory pump, and a valve for regulating the air intake. Of the test material, 100 cc. were used at a temperature of 37 C. (98.6 F.). Each test solution was first whipped for one minute (271 whippings) in a manner which permitted only the air that was taken in through the valve to be mixed with the liquid. Immediately after the whipping, the foam height was measured in cubic centimeters, and again 1, 5, 15, and 30 minutes later.

Protein foam proved stable. Seemingly it plays a more important role in rumen

tympany than is generally believed.

The saponins (which are plant glucosides) also form a stable foam. On the other hand, mucilaginous substances have limited foam stability and they are of no importance in the pathogenesis of frothy bloat. Various foam stabilizers such as bentonite or iron chloride increase the stability of foams. If foam formers and foam stabilizers are used simultaneously, gas expulsion is greatly retarded and this, *in vivo*, results in frothy bloat.

Therapeutic Agents

A large number of physical-chemical experiments were undertaken at the Pharmacological Institute of the Veterinary College in Vienna for the purpose of evaluating by a dependable *in vitro* method the therapeutic value of foam developing bloat remedies. The investigators used as foam formers (1) a 0.05% saponin solution and (2) a 50% potato juice, a protein containing substance, in their foam tester. The defoamer to be tested was added to the whipped, frothy solution in the cylinder in quantities of 0.4 cc. or as 10% aqueous solution or emulsion in amounts of 4 cc. (Water has no defoaming properties.) The reading was taken immediately and again after 1, 5, 15, and 30 minutes. The reading in cubic centimeters immediately after whipping, and before the addition of the defoamer, was expressed as 100% (standard). Then factors were introduced to give consideration to both speed and degree of the defoaming action of the "bloat" remedy (defoamer); the theoretical (maximum) defoaming value was set at 400. From this figure was deducted the sum of the percentages of the destroyed foam multiplied by 4 for the first reading interval (one minute), by 3 for the second interval (five minutes), by 2 (after 15 minutes), and by 1 (after 30 minutes).

The results of these experiments are most interesting when the average of both the saponin and potato juice defoaming values are viewed in order of efficiency (theoretical maximum value 400).

Some drugs contained in the list of average defoaming values are presented here in descending order for purposes of comparison: Rumonal (Haver-Lockhart) — 336.1; cresol — 310.7; potash soap — 278.2; benzene — 229.1; tannic acid — 219.7; oil of turpentine — 205.3; linolic acid — 152.2; petroleum — 149.7; resorcin — 138.4; ethyl alcohol — 122.5; codliver oil — 118.5; peanut oil — 100.3; oleic acid — 95.9; linseed oil — 72.7; white mineral oil — 68.9; glycerin — 64.1; sodium chloride — 49.8.

This list illustrates that many so-called defoamers (either compounded preparations or raw drugs) are of little therapeutic value, and the comparative tests allotted them low defoaming values. The combination of two or more low-valued ingredients does not make a good defoaming agent.

Prophylaxis

The last part of this investigation dealt with prophylactic measures. The investigators tested 73 pharmaceutical preparations and raw drugs contained in their defoaming values list as to their prophylactic properties. In these experiments, they used the foam residues left after evaluation tests were made with saponin or potato juice solutions in the cylinder of their testing equipment (briefly described above); the residues were completely defoamed mechanically and then again whipped for one minute. The new foam height was read in cubic centimeters and calculated in percent of the original foam height of the test substance.

The pure silicone antifoam emulsion previously mentioned proved an excellent prophylactic. No foam was formed in saponin solution. Milk gave 7.8% foam; linseed oil — 25%; oil of turpentine — 74.6%, et cetera. Petroleum, resorcin, skim milk, sodium chloride, sodium thiosulphate, and tartar emetic were valueless (100% foam formation). In potato protein solution, few agents showed prophylactic properties. — *Rudolph Seiden, Ch.E., D. Sc., Kansas City, Mo.*

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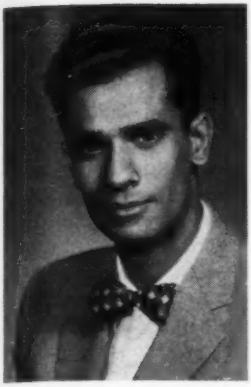

Selye's stress concepts are based largely on hormonal influences. In this Doctor Sinha demonstrates that stress may be largely independent of hormone activity and result directly from environmental influences in the host-parasite relationship, i.e. *alteration in the pathogenicity of the parasite, resistence of the host, and other possible alterations. This discussion was a part of the presentation at the Stress of Livestock Conference which convened at Kansas City, Missouri on December 10, 1957 under the sponsorship of Jensen-Salsbery Laboratories.*

Stress and Host-Parasite Relationship

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ON the biological standpoint, stress is basically a physiological response and not a pathological condition. The host under the influence of stress tries to maintain normal life processes and re-establish functional activities. Poor management, genetic, psychic, and environmental factors are important considerations in this response. Malnutrition, hormone imbalance and fatigue are stress factors having disease producing potential. These effect an abnormal adaptive response by the animal to the inciting pathogenic agent.

Poor management, for example, crowded quarters, lack of adequate shade and water, filthy pens, poor ventilation, abuse and rough treatment, and lack of proper sanitation can produce lowered host vitality. There are many instances of differences in susceptibility or resistance to an infectious disease in varieties or strains of animals. For example, the relative resistance of Algerian sheep to anthrax is well known and inbred Berkshire swine have been found to be highly resistant to brucellosis. Little is known about the psychobiology of animals in relation to stress; however, man's close association with domesticated animals indicate that when life circumstances are adverse, animals also suffer mental distress. The state of nutrition and an hormonal imbalance has been extensively studied by many workers on the standpoint of host-parasite relationship.

Domestic animals are a product of evolution. The animal host is exposed to assault by other living forms that aim to invade as parasites or to destroy. With an awareness of the importance of the direct effects of environmental stress, emphasis on this discussion has been placed upon those alterations within the host that stem from exposure to sudden and relatively rapid changes of many varieties.

Influence on Susceptibility

Resistance or susceptibility of a host to one parasite and not another has always been a challenge to fundamental research. It has been known for many years that environmental temperatures, relative humidities and seasonal variations play an important role in influencing the host-parasite relationship. That is why the greatest incidence of fowl pox among chickens occurs during fall and winter[1]; Japanese B encephalitis arises in epidemic form during the warmest season of the year[2]; epidemics of poliomyelitis in children recur with considerable regularity during the summer months[3]; influenza outbreaks occur during winter and spring seasons in tropics, fall and winter in temperate climate[4]; increase in the incidence of mumps in man is usually recorded during the winter and early spring months[5]; contagious ecthyma of sheep is found frequently during the spring and summer months in the western parts of the United States[6] and epidemic tremors in chickens occurs during winter and spring months of the year.[7] With a better insight into the extent and the mechanism of climatic influences on the infectious process it may be possible to bring more diseases under control.

Increased susceptibility as a result of stress caused by effects of low environmental temperature was first observed by Pasteur, who found that although chickens are normally resistant to anthrax organisms, reduction of their body temperature by chilling made them susceptible. Subsequent work indicates that factors in addition to body temperature are involved in this decreased resistance. Obviously, *B. anthracis* will grow at fowl temperatures, as Pasteur himself demonstrated. True virulence was lost eventually by cultures grown at these temperatures but only after a period far exceeding that required by the organism to kill the chicken.

Treponema pallidum in rabbits maintained at 30 to 35 C. will fail to infect; however, when the environmental temperature is lowered, body temperature is below normal and infection occurs.[8] Muschenheim *et al.*[9] observed the effect of rabbits of induced hypothermia upon experimental avirulent type III pneumococcus infection. Hypothermia resulted in rapid bacteremia and death.

Ispen[10] immunized mice with single doses of tetanus toxoid (5-20 flocculation units) and divided into groups which were maintained for 14 days at respectively 6 C., 25 C., and 35 C. Subsequently the animals were all transferred to room temperature and challenged with 20 lethal doses of tetanus toxin. Highest immunity was shown in the mice submitted to warmer temperature during immunization, the difference being most distinct with a low dose of toxoid.

Moragues and Pinkerton[11] studied the variation in morbidity and mortality rate from a rickettsial infection in mice at various environmental temperatures. Mice were inoculated intraperitoneally with murine typhus rickettsiae and kept at temperatures of 18 to 23 C. and 29 to 37 C. All mice died with a severe peritonitis at the low temperature; whereas the mortality was only 25% in the upper range.

With protozoan infection, Sheppe and Adams[13] observed that when mice were subjected to stress in the form of partial starvation and low environmental temperature following infection with the "non-pathogenic" *Trypanosoma duttoni*, starved mice subjected to low temperatures died much earlier than did those kept at room temperature.

In viral infections, Lillie and coworkers[12] observed a greater intensity of brain reaction in mice to St. Louis encephalitis virus during the winter months. Thompson[14] also studied the influence of temperature on rabbits to infectious fibroma virus. He inoculated fibroma virus into the skin, subcutaneous tissues, testicle and other deeper parts of the body. Tumor like growths observed at the site of inoculation but not in the deeper parts of the body which suggested that fibroma virus required a relatively low temperature for multiplication. In case of spontaneous tumors such as mammary neoplasia, Fuller *et al.*[15] observed among several thousand white mice, a tumor incidence as high as 10 to 15% among those kept in the cold room; while

not a single tumor occurred among those kept in the warm room.

In the classic experiment on poliomyelitis virus studied in mice, Holtman[16] observed that mice acclimated at 13 C. and held at that temperature after inoculation with poliomyelitis virus never exhibited symptoms in less than 11 days, with 13 days being required to establish 50% mortality. Mice adapted to 22 C. and held at that temperature after injection demonstrated 50% mortality at the end of seven days; and mice acclimated to and held at 32 C. developed signs as early as the fifth day. When the mice acclimated at 13 C. and kept at 22 C. and 32 C. after injection showed greater susceptibility than those allowed to remain at 13 C. In contrast to this, mice originally adapted to 22 C. and 32 C. exhibited a greater resistance to infection as indicated by prolonged incubation period when kept at 13 C.

With vesicular stomatitis virus, Griffin, Hanson, and Brandly[17] found that the severity of infection based on mortality rates of mice inoculated intracerebrally, was significantly lower when acclimated at 8 C. than was the mortality rates if the mice were acclimated to 27 C. or 35 C. Incubation period was prolonged in the mice adapted to the lower temperature.

Recently, Sinha *et al.*[18] showed that the severity of Newcastle disease varied in chickens (5 to 6 weeks of age) acclimated to different environmental temperatures. The mortality rate was 100% at moderately high temperatures (85 to 90 F.), 95% at optimum temperatures (70 to 75 F.), 75% at moderately low temperatures (50 to 55 F.), and only 55% at low temperatures (32 to 35 F.). The incubation period was shortest at warmer temperatures and longest at colder temperatures. The nervous signs of the disease was predominant at the warmer temperatures; at the colder temperatures, the respiratory symptoms were more pronounced. The above examples indicated that the environmental temperature higher than optimum can induce stress in host and thereby the animal became more susceptible to a parasite.

The effect of relative humidity may also play an important role with the environmental temperature with regard to host-parasite reaction. Loosli *et al.*[19] observed that humidity in air influenced the survival of influenza A, PR 8 strain. They exposed mice in a chamber at 80 to 90% relative humidity. They found that the virus was no longer infective to mice after one hour; whereas the mice exposed to 10 to 30 minutes following spraying of the virus died with extensive consolidation of the lungs. At relative humidity of 45 to 55% in the atmosphere the virus remained infective for six hours and was lethal for mice up to one hour and ten minutes. At low humidity (17 to 24%), a similar amount of virus in the air produced pneumonia in all exposed mice for at least 24 hours.

By atomizing pneumococci, streptococci and staphylococci in an experimental chamber, Dunklin and Puck[20] showed the rapid death of these infectious agents at humidities of 45 to 55% and much slower rate of death at higher and lower humidities.

TABLE 1.

Calf no.	Inoculum	Clinical signs	Postmortem findings
12	Type A (strain 7707)	Temperature rise (108 F.) 48 hours after inoc.; off feed	Sacrificed after 21 days; organs appeared normal; slight pulmonary congestion
13	Type B (strain 8-88)	No rise of temperature; off feed for 24 hours after inoculation	Sacrificed same day; organs appeared normal
14	Type C (strain 6535)	No rise of temperature; off feed for 24 hours after inoculation	Sacrificed same day; organs appeared normal
15	Types A, B, & C (all 3 strains)	Severe diarrhea; off feed; temperature rise (104.8 F.); nasal discharge	Sacrificed same day; pronounced congestion of lungs; other organs normal

Jungherr[21] observed that Newcastle disease virus was not killed in 55 days at 22 C. with a relative humidity of 7% on sterile burlap and on non-sterile burlap for 22 days at 1 to 7 C. with a relative humidity for 45%.

Seasonal Incidence

During a year, seasonal variation in an infectious disease has been observed in an animal population. On the basis of a large number of outbreaks reported in the various districts of the State of Madhya Pradash in India, Kaushash[22] observed during a one-year period the incidence of main contagious diseases of cattle such as rinderpest, hemorrhagic septicemia, blackleg, anthrax, and foot-and-mouth disease. According to his report, the incidence of rinderpest is much higher in March and later in the month of September, whereas the number of outbreaks are few in November and December. The peak period in hemorrhagic septecemia was noted during the months of July and August and the case reports at its lowest during winter (November to February).

In the case of blackleg, incidence will rise during July and August, similar to hemorrhagic septicemia and then the number of outbreaks will drop during winter months. With anthrax, rainy months (June to August) are responsible for its greater incidence in cattle population. With regard to foot-and-mouth disease, the report indicated over 13,000 outbreaks each month during February and March (early spring) and the disease quickly reaches its low level in May and June (summer).

With regard to this conference on stress effects on livestock industry, the writer wishes to report recent experimental data on pasteurellosis in cattle.

Pasteurellosis in Cattle

Pasteurellosis in cattle is well recognized throughout the United States. According to Sinha *et al.*[23] and Carter,[24] *P. multocida* was isolated in a high percentage of cases reported as shipping fever in cattle. Due to lowered vitality in the animal, disease begins in a herd and is likely to spread rapidly into other herds. The febrile and respiratory conditions are characteristic, and usually associated with shipping. However, carrier animals brought into the herd from livestock sales centers have caused virulent outbreaks among stock not previously exposed to shipping stress.

In our studies, attempts have been made to reproduce pasteurellosis in six- to seven-month-old calves under various experimental conditions.[25] Three serotypes A, B, and C of *P. multocida* were used in these experiments. Strain 7707 (type A) and strain 6535 (type C) were of bovine origin and strain 8-88 (type B) of buffalo origin were maintained at the repository of Jensen-Salsbery Laboratories. The organisms were grown in tryptocase soy broth with 1% rabbit serum for 18 hours at 37 C. Mice weighing 15 to 18 grams were inoculated intraperitoneally with 0.5 ml. of the cultures. Strain 8-88 produced death in mice nine of ten; strain 7707 — eight of ten and strain 6535 — eight out of ten within 24 hours following inoculation.

TABLE 2.

Calf no.	Inoculum	Clinical signs	Postmortem findings
21	Type A (strain 7707)	Temperature rise (104 F.) on the 2nd day to 6th day after inoculation; off feed	Sacrificed after 21 days; emaciated; moderate congestion of lungs; other organs normal
22	Type B (strain 8-88)	Temperature rise (105.4 F.) for 5 days after inoculation; off feed and emaciated	Sacrificed after 21 days; severe lung congestion
23	Types A & B (strains 7707 & 8-88)	Temperature rise (106.8 F.), off feed, diarrhea, severe loss of weight	Sacrificed after 21 days; highly congested lungs; tracheal exudate
24	Control no inoculum	Off feed for 2 days; no rise of temperature	Sacrificed after 21 days; organs appeared normal

TABLE 3.

Calf no.	Inoculum	Clinical signs	Postmortem findings
26 27	Received aerosol exposure of 3 types	Loss of weight; no significant rise of temperature; off feed	Sacrificed; moderate congestion of lungs
28 29	Received cortisone only 0.5 gm. IM	No rise of temperature; no other clinical signs	Sacrificed; organs normal; slight dehydration
30 31	Received cortisone 0.5 gm. IM and aerosol exposure of 3 types	Off feed; diarrhea; temperature rise 103.6 F., rapid and labored breathing; died 21 days after exposure	Animals showed extensive congestion in lungs; hemorrhagic foci in spleen; tracheal exudate; marked dehydration

In trial 1, four calves were used to observe signs of pasteurellosis under no environmental stress. The animals were kept in the isolation units at an environmental temperatures of 85 to 90 F. Five ml. of each strain was inoculated intraperitoneally into three different calves and one received 5 ml. of the mixture of three strains. Bacterial plate count indicated that 5 ml. of inoculum contains approximately one billion live organisms. The results were recorded in table 1.

In trial 2, four calves were used to observe the signs of pasteurellosis following severe environmental stress. Prior to inoculation, the animals were subjected to 150 miles of travel in a truck and after arrival they were exercised for one-half hour. At that time the animals exhibited marked signs of exhaustion and rapid breathing. Then they were showered with cold water for two minutes. The same amount of inoculum of *P. multocida* was used in this trial. The results have been tabulated in table 2.

In trial 3, four calves were used to observe the conditions following aerosol exposure of *P. multocida*. A mixture of strains 7707, 8-88, and 6535 in physiological saline suspension was atomized and each animal received aerosol exposure for 45 seconds. Two of four calves also received intramuscularly 0.5 gm. cortisone. Two calves were kept as controls and received only cortisone (0.5 gm.) intramuscularly. The results are shown in table 3.

The results of this experiment in three trials indicate that two calves died after three weeks when they received aerosol exposure of *P. multocida* and a simultaneous injection of cortisone. Deaths were not observed when Pasteurella strains alone were inoculated into six- to seven-month-old calves under various environmental conditions.

Summary

P. multocida has been isolated from apparently normal animals exhibiting no clinical signs of pasteurellosis. However, it may be logical to think that the organism may alter in its pathogenicity and become virulent within the host when such host is subjected to various stress factors.

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Continued on page 276

Strongyloidiasis in Lambs

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ONE of the writers (Turner[1]) reported that fatal infections of *Strongyloides papillosus* were experimentally induced in otherwise parasite-free lambs by single cutaneous applications of 100,000 or more infective larvae of this parasite. However, we have been unable to find in the literature any record of naturally acquired fatal strongyloidiasis in sheep. The purpose of this note is to report an outbreak of strongyloidiasis in lambs on pasture, during the course of which a few fatalities occurred.

In October 1955, we examined for parasitism a flock of 165 lambs, eight to nine months of age, maintained on a 25-acre pasture in central Maryland. When first observed by us, the lambs were emaciated and many were passing fluid, or soft, stools. Fecal egg counts of *S. papillosus* ranging from 6,480 to 54,000 per gram (av. 14,279) revealed that 14 of the 20 lambs examined harbored moderate to heavy infections; comparatively few eggs of other roundworms were present. One week later, three lambs, which had not been examined previously, died; fecal samples from these lambs contained 61,200, 46,800, and 15,600 eggs of *S. papillosus* per gram, and adult *S. papillosus* recovered at autopsy were 26,000, 23,400, and 9,000 respectively. Other roundworms were present only in insignificant numbers. A severe catarrhal enteritis, which was confined to the upper extremity of the small intestine and closely resembled that previously observed[1] in experimentally infected lambs, was seen in these three fatal cases. The lungs and other internal organs were

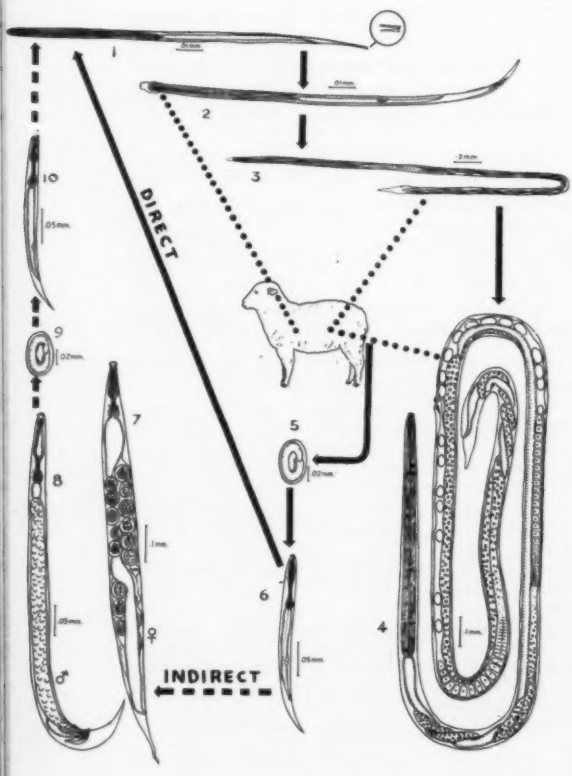

Fig. 1. Schematic diagram of the life cycle of *Strongyloides papillosus* (Wedl, 1856). (1) Third stage of infective larva. (2) Late third-stage parasitic larva from lungs of host. (3) Fourth-stage parasitic larva from intestine of host. (4) Parasitic adult female from the intestine. (5) Egg from feces of host. (6) Newly hatched first-stage larva. (7) Free-living adult female of indirect cycle. (8) Free living adult male of indirect cycle. (9) Egg of free-living generation. (10) Newly hatched first-stage larva of indirect cycle. (Drawn by J. C. Hwang after Basir.[8])

normal. The remainder of the flock was transferred to a new pasture a few days later. No further deaths occurred, and the lambs were in good health five weeks later.

These data incriminated *S. papillosus* as the primary etiologic agent in this epizootic and indicated that under certain conditions naturally acquired *S. papillosus* infections can cause acute clinical effects and death, thus confirming the experimental findings previously reported.[1]

Although this flock had received regular anthelmintic treatments with phenothiazine, the drug apparently was of little value in controlling this parasite. Poor nutrition, resulting from inadequate forage, probably was an important factor. The lambs had to be fed supplemental roughage in racks, as the pasture deteriorated from overgrazing. Rees[2] has reported that sheep on improved pasture had fewer *S. papillosus* than sheep maintained on unimproved pasture; her findings were suggestive of a direct relationship between the quality of forage, the health of the animals, and their resistance to infection. Because the outbreak of strongyloidiasis reported herein occurred in October, the development and survival of relatively large numbers of infective larvae on the pasture with the advent of moderate, early autumn weather conditions may have been another factor in its causation. Substantial accumulation of larvae on the pasture during the summer seems less likely in view of the report by Doll and Hull[3] that larvae of this parasite did not survive well on pasture in Kentucky during the summer. Infective larvae (fig. 1-1) of this parasite are not enclosed in the protective sheath common to most nematode larvae infecting ruminants and are, therefore, quite susceptible to certain climatic conditions to which many ensheathed larvae are resistant. Early fall weather conditions, characterized by moderate mean temperatures and adequate rainfall, in central Maryland are generally favorable for the development of infective larvae of *S. papillosus*, and in 1955 apparently favored the accumulation of viable larvae in numbers sufficient to cause this outbreak.

Probably three factors were mainly responsible for the termination of this outbreak: (1) The gradual development in the lambs of an acquired resistance to this parasite, such as one of us (Turner[4]) has shown to occur in experimentally infected lambs exposed to 10,000 to 30,000 infective larvae at two day intervals for 20 days, (2) the transfer of the flock to a better pasture, and (3) less favorable conditions for the development of infective larvae as the fall season advanced and temperatures became lower.

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Reservoirs of Bluetongue Virus

Bluetongue of sheep (formerly termed "sore muzzle" in sections of southwestern United States) almost always disappears in all American states where freezing weather occurs. During certain months of the year, vectors in the form of small insects, which are essential for the transmission of the causative virus, are inactivated. Therefore, in those states where winter grazing of wheat is the principle form of forage for sheep the disease is relatively uncommon. However, since cattle are said to retain the virus of bluetongue for much longer than sheep without exhibiting clinical signs of infection, these animals can serve as virus reservoirs until the following warmer insect season. This means that strict quarantine should be maintained to hold infection under control.

The authors of this article have pooled their knowledge with the thought that differing approaches to this problem by the physician and the veterinarian might lead to some definite conclusions. Some of these might serve as a basis for field experimentation by practitioners who can prove or disprove their validity.

The Ketosis Problem

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THE ketosis problem in dairy cattle has plagued the veterinary profession for a long time. Literature concerning the problem is voluminous and confusing, although certain facts are well recognized. Some authors consider ketosis to be a specific disease, whereas others insist that it may occur as a symptom of various conditions. It is observed most frequently in high producing dairy cows. It may occur in association with starvation, high fat diets, highly acid feed (ensilage), acidosis, impaired liver function, infectious diseases and endocrine disorders. A form of starvation ketosis frequently precedes shipping fever. Irregular feeding during transit causes starvation, and the excitement increases thyroid activity. The increase of thyroid activity induces a breakdown of body protein. Hyperthyroidism is a disease of protein metabolism and breakdown of body protein always results in fever. Pasteurella bacteremia is a concomitant symptom.

Udall[10] reported that "what appears to have been ketosis" was described in 1849. Sjollema and van der Zande[14] described the clinical syndrome and reported acetone bodies in blood and urine. Stinson[15] found beneficial therapeutic results by giving injections of glucose subcutaneously.

Dye and McCandless[8] claim that no connection exists between the oxidation of carbohydrates and fats. They state that whenever the oxidation of sugars increases, oxidation of fats is decreased, and *vice versa*. If this were so, there would be no ketosis problem. The reason is that sugars being low, the animal would be forced to utilize more fats, but this is not the case. The statement contradicts the universally accepted law of physiology. Sampson, Gonzaga and Hayden[9] found normal blood sugar levels in cows suffering from ketosis whereas Fraser *et al.*[4] stress the fact that they found marked hypoglycemia in sheep which exhibited no symptoms of ketosis. As to ketobodies in the blood, Shaw[11] reported ketosis established clinically without

blood ketones being above normal, whereas Johnson[6] and others found the ketobody level invariably high. Shaw[11], on the other hand, reported no visible symptoms of ketosis in a series of starvation experiments while blood levels of ketones were as high as in clinical ketosis. Various authors have reported low as well as normal alkali reserves.

All these findings have been applied to prove or disprove certain concepts of ketosis. Ketosis, as to the pathologic physiology of blood, means the disturbed relationship between ketobodies and carbohydrate plus alkali. The absolute levels of the substances concerned have no clinical significance except in relation to each other. Authors writing on ketosis have seldom correlated all three factors; one or two of the factors concerned are universally neglected. It is possible to report in starvation ketosis the finding of increased levels of ketobodies with no manifestation or clinical signs because the buffering effect of a normal alkali reserve counterbalances the higher ketobody level. High doses of ammonium chloride, given to several control animals at the end of the dry period and before the occurrence of starvation, should indicate what happens when the alkali reserve has been reduced. Although this experiment has not been performed, it is, in our opinion, the only approach to reproducing ketosis experimentally.

Controversial Conclusions

Other contradictory statements might be quoted: Johnson[6] states that therapy with alkali salts should be avoided because it does lead to alkalosis (why he objects to this in acidosis is not clear). Sampson[5] quotes that alkalosis does have a ketogenic effect. Johnson[6] contradicts himself by stating that propionic acid acts as an antiketogenic agent, and that the same acid when given by stomach tube causes acidosis. We must note the contradictory statements that alkalies as well as acids, act as ketogenic agents, and that propionic acid is both ketogenic and antiketogenic. The treatment of ketosis with propionic acid compounds is contraindicted because the liver is unable to resynthesize dextrose and glycogen from propionic acid while the animal is suffering from acidosis.

To confuse the veterinary practitioner further, "stress" has been used to explain ketosis. The stress complex might comprise a certain symptomatology but does not say anything about the etiology of a given clinical picture. We can better define the adaptation syndrome by calling it a drop in the cholinesterase level in the blood, perhaps aggravated by increased viscosity of the latter.

The fact that cattle for generations have been bred for higher and higher milk yields without a corresponding increase in the electrolyte content of the blood is the cause of ketosis. When ketosis develops, hypoglycemia and ketonemia usually exist. Veterinary practitioners recognize these conditions and observe in addition that inappetence, decreased milk production, incoordination, marked loss of weight and severe dehydration are part of the syndrome.

Etiology

It is our contention that bovine ketosis is not only an imbalance of glucose and volatile acids, but is also due to a lowered alkali reserve.

Glucose Requirements

The lactating mammary gland requires tremendous amounts of glucose. Shaw *et al.*[10] calculated that a cow producing 25 kg. (55 lb.) of milk daily will require more than 1,000 gm. of glucose. Roberts and Dye[7] estimated that a 1,100 lb. cow should require about 1,500 gm. of glucose daily. The large glucose utilization by the mammary gland was not considered in this calculation. The glucose requirements of other tissues of the cow are probably different from those of non-ruminants because of relatively large amounts of acetate and butyrate available. This may be expected to have a marked sparing effect on glucose utilization. However, this function is greatly impaired in the animal with ketosis.

Alkali Reserve

Calcium phosphate and potassium salts are plentiful in milk. The sudden demand for these elements in a fresh cow can result in low levels of these compounds.

Acidosis is a frequent symptom in bovine ketosis, and may be due to a low alkali reserve as well as to excessive amounts of acids to be buffered. Diuresis occurs because base and water must be wasted in excretion of excessive acid. Therefore, dehydration is a frequent result of acidosis. The excessive thirst seen in acidosis also increases the loss of electrolyte through the kidneys because of the doubled intake of water.

Ketone Bodies

Carbohydrate fermentation in a cow results in the volatile fatty acids: Acetic, propionic and butyric. Propionic acid is a source for glycogen. Shaw[10] estimates that the healthy cow may obtain an excess of 800 gm. of glucose from propionic acid per day. Since the resynthesis of glycogen from propionic acid by the liver is impaired in the animal with ketosis, glycogen must then be obtained from protein.

The liver forms ketone bodies from fat. They accumulate in excessive amounts when carbohydrate metabolism is reduced or disturbed. As acetone is being formed, beta-hydroxybutyric acid and acetic acid will bind bicarbonates, producing a state of acidosis.

Dextrose Administration

Usually 500 ml. of a 50% dextrose solution is administered intravenously to the cow with ketosis. This provides an immediately available supply of glucose which is transitory. Shaw[11] showed that 20 gm. of glucose per hundredweight injected intravenously in normal and ketotic cows produced a similar increase in blood glucose levels up to 120 mg. per 100 ml., but within 80 minutes these values approached the starting levels. In addition the renal threshold for glucose is 100 mg. per 100 ml.[1]; therefore, intravenous glucose has little chance to persist in the blood stream.

In absorption studies, it was found that orally administered dextrose was not absorbed readily from the rumen. Bell and Jones[1] reported that oral administration of sugar or dextrose to ten cows in doses up to 8 gm. per kg. of body weight did not produce variations in blood glucose outside the range of normal fluctuations. Shaw *et al.*[13] further emphasized this low absorption of glucose from the rumen, but suggested that unusually large amounts of dextrose might be absorbed. However, when Hungate *et al.*[8] administered 3 lb. of glucose in 2 liters of water (70%) directly into the rumen of a sheep, the animal died. This high a concentration should be avoided when glucose solutions are administered by stomach tube. When the animal starts to recover, tube feeding can be discontinued, and glucose can be added to the drinking water.

All of these observations were based on normal animals with normal fermentation. In the normal animal the rumen is filled with fermented and hydrolysed cellulose which swells to many times its previous volume after being subjected to the enzymatic action of bacteria. When glucose in solution is added to a full rumen most of it is absorbed by the spongy cellulose, and therefore little sugar can come in contact with the rumen wall to enter the bloodstream directly.

On the basis of our own studies, we believe that simultaneous administration of dextrose and insulin will enhance the value of dextrose. Insulin increases glycogen storage in the liver so that the injected glucose will be available for longer periods. This practice is well established in human medicine and practitioners can deduce the value of the combined application of dextrose plus insulin in clinical cases.

In a herd of normal cows, levels of sugar in the blood were determined by the photoelectric method. One 500-unit dose of insulin was administered intravenously. At 1 and 1½ hours, blood sugar determinations showed a decrease. After the last sampling, 4 lb. of dextrose was given by stomach tube. Ten and 30 minutes later, blood-sugar levels were again approximately

TABLE 1.

Cow	Fasted (hr.)	Blood Sugar (normal) (mg./100 ml.)	Insulin I.V. (units)	Blood sugar after 1 hr. (mg./100 ml.)	Blood sugar after 1½ hr. (mg./100 ml.)	Dextrose orally (lb.)	Blood sugar after 10 min. mg./100 ml.	Blood sugar after 30 min. mg./100 ml.
1	3	74	500	48	46	4	70	77
2	24	69	500	46	47	4	96	142

normal. Similar results were obtained repeatedly. Cows were then starved, and insulin and dextrose given as before. At 10 and 30 minutes after oral dextrose administration, blood sugar had risen to high levels. This also was repeated many times with the same results. Typical findings are tabulated in table 1.

The comparison proves that rapid absorption of dextrose takes place from the emptied rumen and that rumen walls are not impermeable as has been stated by some authors. This would indicate that oral administration of dextrose should be beneficial in ketotic cows. The assertion by Dye and McCandless that all carbohydrates are converted into volatile acids and that feeding dextrose solutions to ketotic animals will only tend to increase the volatile acids in the blood does not hold true for starved and ketotic animals. The absorption of dextrose is so rapid that there is not time enough for the rumen bacteria to ferment the small molecule.

Reduction of Volatile Acids in the Rumen

Cattle have two sources of acids. They are the conversion of glucose and starch to the volatile acids, acetic, propionic and butyric in the rumen, and the oxidation of fat by the liver.

It is possible that 30 gm. or more of magnesium peroxide, administered orally twice daily until the cow returns to normal, may prevent the conversion of sugar, starches and cellulose in the rumen by anaerobic bacteria. Peroxide will stop catalytically the fermentation by the anaerobic flora, and thus dextrose will be made available directly. Magnesium peroxide will destroy all anaerobic bacteria temporarily and, while being reduced to magnesium oxide, will serve as a further adjuvant in the treatment of ketosis.

Hormone Therapy

Shaw and coworkers[12] and Dye *et al.*[8] and others have demonstrated the value of cortisone and ACTH in the treatment of bovine ketosis. The value of this treatment is recognized, but other therapeutic approaches are worthy of consideration because of the cost of this therapy.

Prevention of Ketosis

It is necessary to consider more closely mineral metabolism. Our cultivated soil is being depleted of sodium and the highest values for sodium-saturated soils are found in uncultivated forests. When the soil is taken into cultivation, considerable sodium is lost through leaching and through subsequent crops. When the soil turns acid the farmer thinks of adding lime but seldom, if ever, sodium. For sodium deficiency in cattle, farmers rely upon rock salt as compensation. Sodium chloride is a neutral salt, but cattle, on the other hand, require alkaline salts to combat the steady stream of fatty acids entering the blood. An alkaline mineral-mixture containing 15% sodium chloride, 35% sodium bicarbonate and roughly 50% calcium carbonate is better than rock salt alone. (Trace-elements, phosphorus, cobalt, etc. are omitted and only the sodium:calcium ratio is considered.) Such an alkaline mineral supplement should have 2% sodium fluoride added because fluoride is the only element we know of which induces the liver to store glycogen (a *sine qua non* in basic therapy of ketosis). Furthermore, fluoride causes fluid-retention, thus counteracting dehydration. Fluorine also has a marked effect upon cation retention, causing the body to absorb and store more cations which in turn enhances fluid retention. When administered in small amounts with calcium, sodium fluoride is not toxic. Experiments by the authors

with yeast cultures show that growth is completely arrested when fluorine has been added to the agar. These toxic concentrations of fluorine are neutralized by the presence of calcium. Yeast colonies begin to grow as if no fluorine is present at concentrations of one part of fluorine to 500 parts of calcium.

Discussion and Conclusion

Observations and limited studies indicate that ketosis in dairy cattle is not only an imbalance between glucose and volatile acids, but also a lowered alkali reserve. The restoration of alkali reserves may be aided by the formation of magnesium oxide from orally administered magnesium peroxide which should reduce anaerobic rumen fermentation, thus preventing formation and absorption of volatile acids. While glucose is not absorbed readily from the rumen containing large amounts of cellulose, it is absorbed directly and rapidly from the rumen of the cow fasted for 24 hours. Since insulin increases glycogen storage in the liver, it would appear that it should be a valuable aid in cows treated with dextrose intravenously or orally. Prevention of ketosis might be aided by raising the alkali reserve by feeding alkaline rather than neutral salts, in addition to an adequate supply of calories in the diet.

Although these studies have been limited and the observations made in normal cows, this approach to therapy in the cow with clinical ketosis appears to be justified, and further word should be stimulated in an attempt to solve this problem.

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Laryngotracheitis of Fowl Spread by Indirect Contact

The University of Connecticut has recently reported that one of their research projects indicates that laryngotracheitis of fowl is spread by indirect as well as by direct contact or association of infected and susceptible birds. In the past, direct spread only has been considered to be the primary mode of infection. Now it has been demonstrated that rodents, crows, dogs, man, et cetera, may be mechanical carriers of the contagion. This means then that vaccination, and the strictest sanitary measures must be resorted to if the condition is to be satisfactorily handled.

This is the first report that genital trichomoniasis has been diagnosed in bulls maintained under range conditions. Examination of preputial washings has revealed a surprising infection rate and has been determined to be responsible for reproductive disorders and a significant loss in calf crops.

Trichomoniasis in Range Cattle

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BOVINE genital trichomoniasis is caused by microscopic, flagellated protozoan parasites known as *Trichomonas foetus*. In the cow, infection is usually self-limiting, and because of resistance developed as a result of infection, animals may be protected against reinfection for a time. In the bull, trichomoniasis is usually permanent unless the animal is treated successfully. It is known that some untreated bulls have retained such infections for ten years or even longer.

Genital trichomoniasis occurs chiefly in dairy cattle and in some beef animals maintained on pastures or in feed lots. During the fall of 1954 and the spring of 1955, however, trichomonads were found in preputial washings of eight of 44 beef bulls from ranges in Utah County, Utah. Insofar as the writers are aware, this is the first time that genital trichomoniasis has been diagnosed in beef animals maintained under range conditions. This finding suggested the desirability of more extensive examinations of beef animals from ranges in the area in order to, (1) determine the extent to which genital trichomoniasis occurs in range cattle in the central Rocky Mountain Area, and (2) provide a basis for estimation of production losses caused by this disease in affected herds. Accordingly, examinations for trichomonads were made of 383 beef bulls in eight localities in Utah, Idaho, and Colorado, beginning in the fall of 1954 and continued through the spring of 1956.

In each of the eight localities where examinations were conducted, there is an association of cattle owners. In each association the majority of bulls and cows are maintained on a common national forest range during the grazing season. During the remainder of the year, each member of the association individually provides for his own animals on privately owned pastures and feed lots. In addition, a small number of herd bulls are confined to the various ranches for the breeding of selected pure-

bred cows, some of which are put on the range. These conditions made it difficult or impossible to keep adequate breeding and calving records.

Examination Procedures

Examinations were made soon after bulls were brought in from the ranges in the fall or before they were sent to ranges in the spring. Preputial samples were collected from individual animals by douching with physiologic saline.[1] In animals in which the external surface of the prepuce was dry and clean at the time of examination, preliminary cleansing of the area was omitted. Douchings from each animal were stored separately under refrigeration and transported to the laboratory for examination, which was usually done within five hours of collection.

Each sample was centrifuged, and a portion of the sediment examined microscopically. The remainder was inoculated into a tube of sterile culture medium. The medium used was a modification of Plastridge's beef extract medium, which was designed specifically for culturing trichomonads.[2] Cultures were examined for the presence of these organisms after incubation for four or five days at a temperature of 37 C. Early in the investigation, it was observed that microscopic examination of centrifuged samples failed to reveal the presence of trichomonads in a considerable number of samples which were later found to be positive by the culture technic. Consequently, direct microscopic examination was discontinued during the latter part of the work, and diagnosis was based on the presence or absence of organisms in the cultures.

Findings

Results of examinations made on 383 beef bulls from associations in Utah and adjacent States, are summarized in table 1. Five associations are located in Utah, two in Idaho, and one in Colorado. In each of the first four listed in the table, all bulls were examined; in the remaining four, only a portion of the animals were included in examinations. Trichomoniasis, as determined by the presence of *T. foetus* in preputial washings, was diagnosed in four of the eight localities. The proportion of infected animals in the different areas ranged from 5.6 to 18.2% of those examined (table 1). In the locality first listed (L) all infected bulls had been on the range during the summer preceding examinations. In each of the other localities, some infected bulls had never been on the range, but had been used to breed heifers and cows which were then sent to the range. The number of females per bull on these ranges varied from about 30 to 50 in different associations. No examinations were made of heifers or cows.

Of the 23 bulls found to be infected, 12 were Shorthorns, eight were Herefords, two were Beefmasters, and one a Milking Shorthorn. The predominant breed of the bulls examined was Hereford. The large proportion of Shorthorns found to be infected suggests the possibility of a rather high susceptibility to these parasites in animals of this breed. No definite conclusions concerning this point may be drawn at this time, however, because of the relatively small numbers involved and because of a lack of uniformity in breeding histories of the bulls in question.

As a control measure, bulls found to be harboring trichomonads were either treated or sent to slaughter. Those selected for treatment were transported to the Veterinary Science Department of the Utah State University, where treatments were conducted on an experimental basis. In all, 16 of the 23 bulls found to be harboring trichomonads were treated; the remainder were sent to slaughter. Three of the 16 animals were purchased by the Utah Agricultural Experiment Station for experimental purposes prior to treatment. The remaining 13 were returned to the owners after treatment was completed and the animals adjudged to be free of trichomonads, as determined by repeated failure to find these parasites in preputial washings. That trichomoniasis was effectively controlled in one association (L) is indicated by the finding of no infected bulls on examinations in each of the two years following the one

TABLE 1. Summarizing the Occurrence of Genital Trichomoniasis (*Trichomonas foetus*) in Beef Bulls of Eight Cattle Associations in Utah, Idaho, and Colorado, 1954-1956

Association and locality	No. examined	No. infected	% infected
L Utah	44	8	18.2
SR Utah	56	4	7.1
SP Utah	142	8	5.6
P Utah	13	0	....
H Utah	18	0	....
M Idaho	35	3	8.6
P Idaho	30	0	....
G Colorado	45	0	....
Total	383	23	6.0

in which trichomoniasis was discovered and control measures instituted. In this association the calf crop, which was reported to be poor in 1954 and 1955, was improved in 1956.

In view of the high incidence (5.6 to 18.2%) of genital trichomonads in bulls of the four associations where infections were discovered, it was considered desirable to estimate losses in production that could be associated with the presence of this genital disease. Because of the inadequacy of information upon which it must be based, such an estimate can only be an approximation. Considerable variation occurs between individual ranching operations and between cattle associations. Inadequate breeding and calving records and lack of knowledge of the duration of infection, and of feeding costs, complicate the estimating process. An estimate was made, however, of potential losses associated with trichomoniasis in the case of one association (L). Losses that could be attributed to the eight infected bulls in this one association alone were estimated at about $6,200. annually. This amounts to about $800. per bull per year.

If it is considered that this estimate is representative of all of the infected bulls discovered in this survey, the cost of trichomoniasis to the four associations in which the 23 infected animals were found may be in the neighborhood of $18,000. annually.

The method of arriving at these estimates and facts on which they were based are briefly as follows:

Methods of Estimation

1. The ratio of breeding cows to bulls maintained on the range by the association in question (L) is about 35 to 1.

2. Under the conditions prevailing in this area about 75% of breeding females normally produce a calf to weaning age. Consequently, about 26 cows for each bull could normally be expected to conceive and produce a calf each year. Each year about 15% of the total number of breeding females is replaced by younger animals. The proportion of females susceptible to trichomoniasis would be dependent chiefly on the replacement ratio and on the duration of the infection in the herd, because of temporary immunity following infection. Since no information concerning the latter point was available, it was assumed for the purpose of this calculation that about two-thirds of the females would develop trichomoniasis when bred by an infected bull.

3. Assuming that each infected bull would each year breed a full complement of cows, it may be estimated that about 17, or two-thirds, of the females would fail to become pregnant as a result of trichomoniasis.

4. It is known that the duration of initial infections with *T. foetus* is about three months on the average.[3] Consequently, each susceptible cow would experience a delay in conception of about four months (three months for the infection to expire and one additional month for the animal to conceive).

5. For the eight bulls (L) found to have trichomoniasis, each of 136 cows could therefore be expected to experience a delay in conception of about four months.

6. The total cost of maintaining these 136 females during the period of potential nonproductivity engendered by trichomoniasis was estimated at about $1,100.

7. The average growth rate of a healthy calf during the first four months of its life under these conditions was estimated at about 1.75 lb. per day, so that for the 136 potential calves during the four-month period, this would amount to a loss in beef production of 28,600 pounds.

8. If the price of beef is considered to be 18 cents per pound, this would represent a loss of about $5,100.

9. The total loss in beef production and from wasted feed of cows involved can be calculated, therefore, at about $6,200. per year.

Other sources of potential losses, which were not taken into consideration in preparing the estimates herein summarized include those due to pyometra and abortions. In cases of pyometra, females may retain trichomoniasis up to one or two years.[4] In such cases the loss in production would, therefore, be much greater than in cases where pyometra does not occur.

Additional losses, which were not estimated, relate to those resulting from slaughter of infected bulls, as well as to expenses involved in treatment of the remainder.

In view of the aforementioned facts, the estimate of an annual loss of $6,200. per year from trichomoniasis in cattle association (L) is, no doubt, conservative.

Summary

1. A total of 383 beef bulls in eight localities in Utah, Idaho, and Colorado were examined for genital trichomoniasis (*Trichomonas foetus*) during the period 1954 to 1956. Most of these bulls were associated directly or indirectly with range operations.

2. In all, 23 (6%) of animals examined were found to be infected with these parasites. The infected animals occurred in four of the eight localities surveyed.

3. Incidence of trichomonads in bulls in the individual associations ranged from 5.6 to 18.2%.

4. It was estimated that losses in production because of reproductive disorders in cows bred by infected bulls may amount to about $800. annually per infected bull. On this basis the 23 infected bulls discovered by the examinations would cost the owners in the neighborhood of $18,000. each year.

5. The results of the relatively small number of examinations of beef bulls herein reported indicate that bovine genital trichomoniasis may be widespread in range cattle in the central Rocky Mountain area, and probably causes significant losses in the annual calf crop.

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Leptospirosis Carriers

Importance of dogs as carriers of Leptospira infections to man and dogs was emphasized by Dr. Erskine V. Morse of Michigan State University.

Doctor Morse stated that approximately one of three dogs in several metropolitan areas gave serological evidence of leptospirosis. Of 252 veterinarians tested, 52 gave diagnostic test reactions to the disease. In all of these cases, past infection had been diagnosed as another disease or had been so mild that it passed unnoticed. These veterinarians were not aware they had ever been infected or had at any time suffered attack of the disease.

Leptospirosis is still rated as the third most important infectious disease of swine and cattle in the United States. It is responsible for losses of almost $100,000,000. annually.

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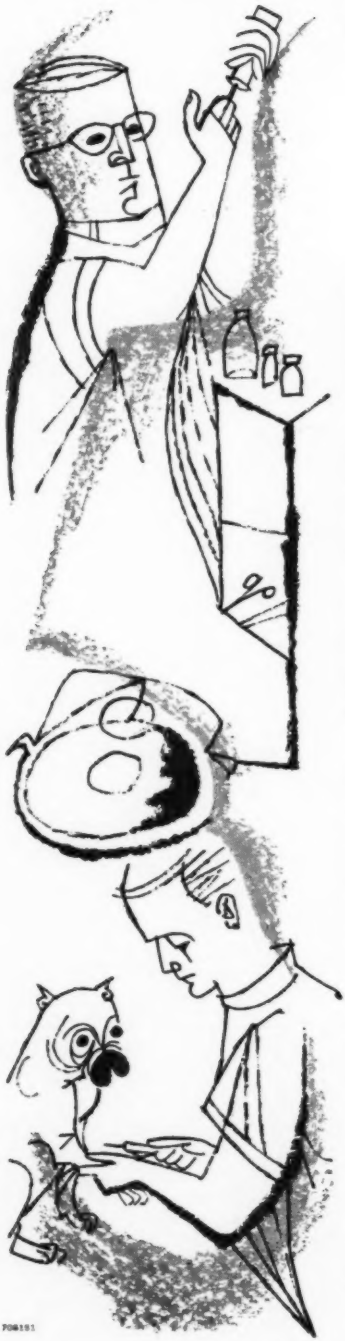

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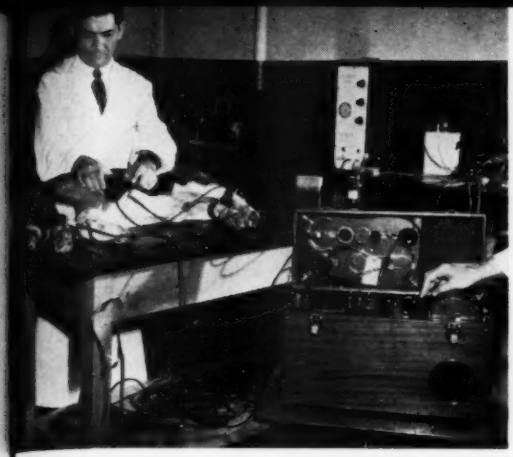

Recording an electrocardiogram and phonocardiogram in the Heart Station of Veterinary Medicine, University of Pennsylvania. All electrocardiograms are taken with the dog in the position illustrated. Three limb lead electrodes are attached to the two forelegs and the left hind leg. A ground lead is attached to the right hind leg. An exploring lead is being held on the left side of the thorax in the sixth intercostal space at the costochondral junction and the microphone is in place at the mitral area. The electrocardiograph is a Sanborn Twin-Beam Cardiette.

Cardiac Arrhythmias, Heart Murmurs and Congestive Heart Failure in Dogs

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THREE aspects of cardiology important in diagnosis and management of heart disease are: A knowledge of the most frequent arrhythmias, an appreciation of the causes of heart murmurs, and an insight into the current concepts regarding congestive heart failure.

Common Cardiac Arrhythmias

Before discussing arrhythmias the structures concerned in the initiation of the heart beat and conduction of the cardiac impulse will be briefly reviewed. Figure 1 shows a schematic version of the specialized tissue of the heart; the sino-atrial node, atrioventricular node, bundle of His, and Purkinje network. The heart beat is initiated in the sino-atrial or S-A node. The impulse spreads over the sheet-like atrial musculature, exciting the muscle fibers which contract, expelling blood from the atria into the ventricles. It is during the spread of this impulse that the P wave is inscribed in the electrocardiogram (figure 2). The impulse reaches and excites the atrioventricular node and, after a delay during which atrial contraction is completed, passes over the bundle of His and is further conducted, *via* the Purkinje network, into the ventricular musculature. During passage of the impulse in the bundle of His, no evidence of electrical activity marking its course is observed in the electrocardiogram. Thus the trace remains at the isopotential position (except for a small deflection owing to atrial repolarization which may be visible in certain leads) from the end of the P wave to the beginning of the QRS complex. The latter marks the rapid spread of the impulse from the terminal portions of the special-

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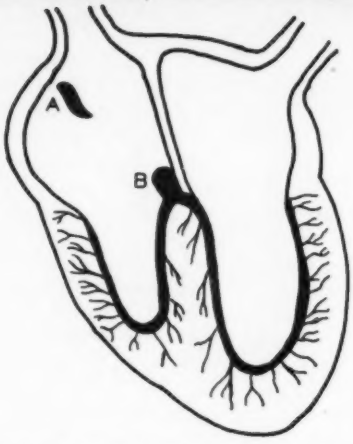


Fig. 1. Schematic drawing of the mammalian heart. A., sino-atrial node and B., atrioventricular node, both located in the right atrium. Two major branches of the bundle of His are seen passing in the subendocardium of the interventricular septum to each ventricle. The Purkinje network is represented by the small twigs branching from these bundle branches.

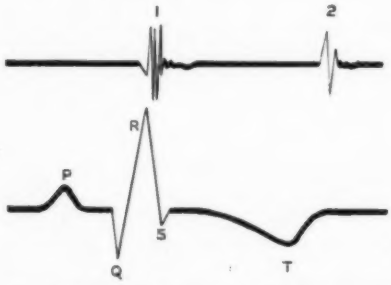


Fig. 2. Schematic diagram of the simultaneously registered phonocardiogram (upper) and electrocardiogram (lead II) of the dog. 1. First heart sound. 2. Second heart sound. P,Q,R,S,T, electrocardiographic waves. The third and fourth heart sounds, which are not normally heard in the dog, are not depicted.

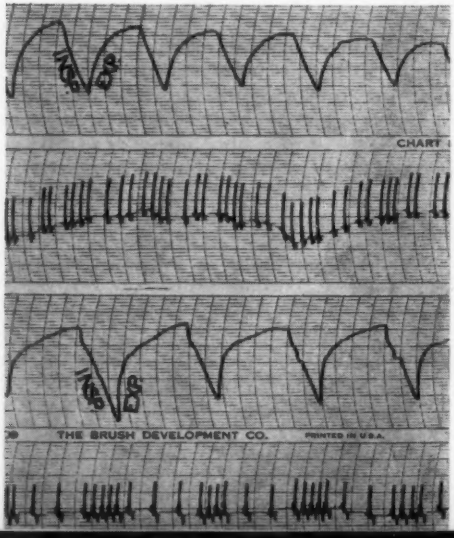


Fig. 3. Pneumogram (upper curve) and electrocardiogram (lower curve) from two resting dogs showing respiratory sinus arrhythmia. (From Detweiler, *Schweiz. Arch.*, 98:475, 1956.)

ized conduction tissue into the ventricular musculature, causing the excited muscle fibers to contract and producing concerted systole of the two ventricles. Following the QRS complex, during the interval when the muscle cells of the ventricular mass are depolarized and in a state of contraction, the electrocardiographic trace again returns to the isopotential position (the RS-T segment). The end of ventricular systole, accompanied by repolarization of the muscle fibers, is reflected in the electrocardiogram by the inscription of the T wave. (See figure 2.)

Heart sounds are depicted in figure 2 which shows their relationship to the electrocardiographic waves. The first heart sound is associated with closure of the atrioventricular valves at the onset of ventricular contraction and the second sound with closure of the semilunar aortic and pulmonic valves at the beginning of diastole. Rarely, a third sound may be detected shortly after the second sound. It occurs at the time of rapid ventricular filling when blood, which has accumulated in the atria and great veins during ventricular systole, rushes into the expanding ventricles. A fourth heart sound, associated with atrial contraction, may be heard if the interval between atrial and ventricular contraction is prolonged or the ventricles fail to contract, as in atrioventricular block. (See figures 8 and 9.)

Sinus Arrhythmia. In dogs at rest the heart rate is periodically accelerated and retarded owing to phasic variations in vagal activity, usually related to the breathing cycle. Figure 3 shows simultaneous pneumograms and electrocardiograms taken from each of two resting dogs. The presence of this arrhythmia is one indication that the animal is in a resting state, not greatly excited, and that the cardiovascular system is not under strain. It disappears under any circumstances (such as excitement, fever, exercise, or certain cardiovascular disturbances) which result in diminution of vagal

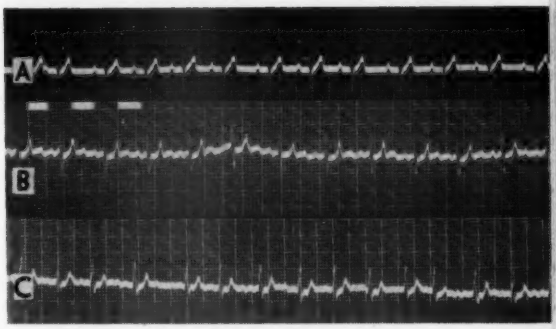


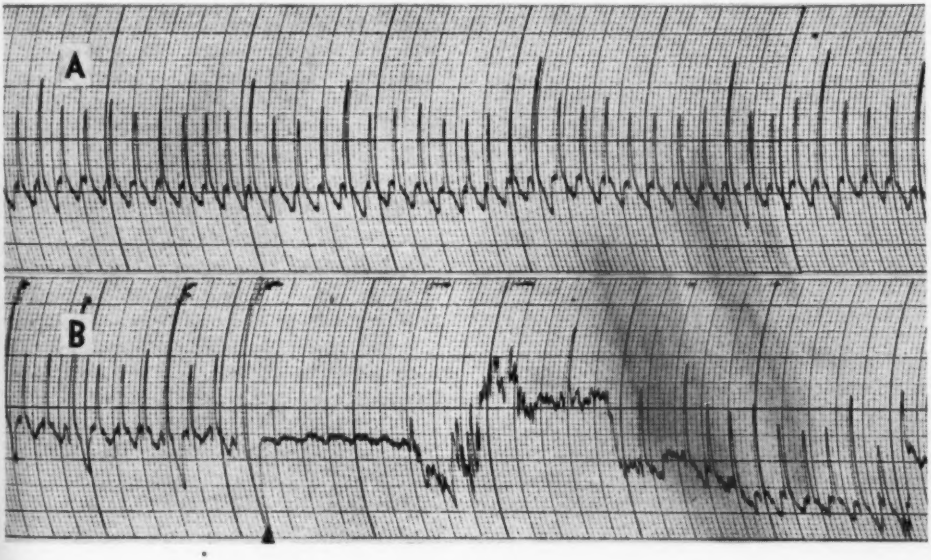
Fig. 4. Sequential development of auricular premature beats, auricular flutter, and auricular fibrillation in a dog with congenital hypoplasia of the right atrioventricular valve. A. Lead III. Electrocardiogram taken November 29, 1949, when the dog was two years of age. The second complex (the first complex is partially hidden by the letter A) is a premature auricular beat. Sinus arrhythmia is also present. B. Lead III. Record taken January 9, 1953, showing auricular flutter (flutter rate 440 per minute) with 4:1 atrioventricular block (ventricular rate 110 per minute). C. Lead II. Electrocardiogram taken September 17, 1954, showing auricular fibrillation (f-wave rate 750 per minute, ventricular rate 128 per minute). (From Detweiler, *Zentralbl. Vet.-Med.*, 4:509, 1957.)

tone. Lange[1] found that sinus arrhythmia was never present in puppies under four weeks of age.

Thus, the absence of sinus arrhythmia at rest in dogs (puppies excepted) may be taken as an indication that the animal is not in a resting or basal state, and is to be expected in cardiovascular disease as well as from any other influence which diminishes the tonic action of the vagus on the heart.

Auricular Flutter. This rare arrhythmia[2] may be the cause of excessive tachycardia. The electrocardiogram is characterized by the absence of P waves and the diagnosis is based on the finding of auricular waves (flutter or F waves), regular in form and rate, which in one or more leads show no isoelectric periods. The rate of impulse formation in the auricles may exceed 400 per minute. Since every auricular impulse may not traverse the atrioventricular node, the ventricular rate may be slower than the auricular. For example, the auricular rate in figure 4 is 440 per minute, but only every fourth impulse is conducted (4:1 atrioventricular block) so that the ventricular rate

Fig. 5. A. Auricular flutter with 1:1 ventricular response (ventricular rate exceeding 343 per minute.) B. Eyeball pressure applied shortly before arrow resulted in transient ventricular standstill (atrioventricular block) and the flutter (F) waves can be seen. The oscillations following the F waves were caused by struggling. (From Detweiler in Hoskins and Lacroix, Canine Medicine, 2nd ed., in press.)



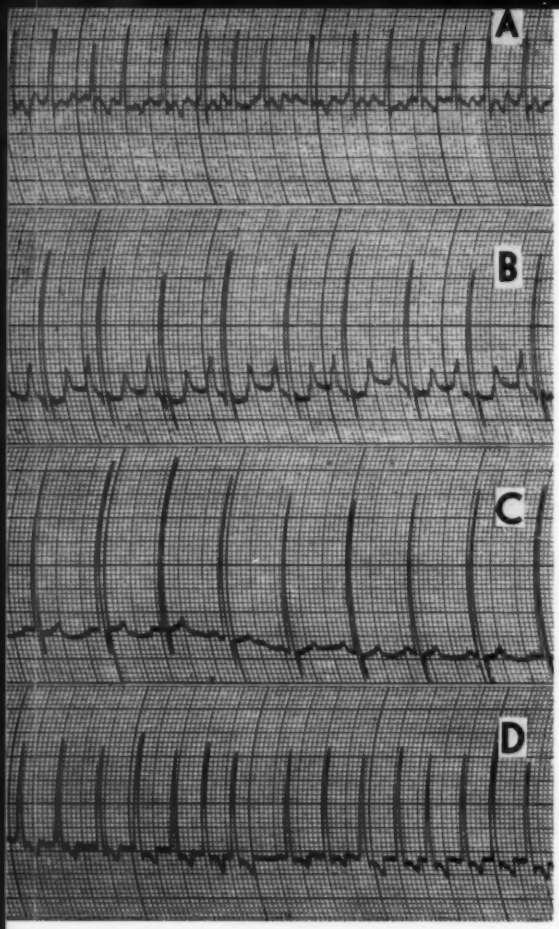


Fig. 6. Auricular fibrillation in a dog with patent ductus arteriðsus, abolished with quinidine sulfate. A. Auricular fibrillation. B. Record taken on the morning following a course of quinidine sulfate. Note the high, slurred P waves. C. An electrocardiogram taken one month later. The P waves now appear more normal. D. Spontaneous return of auricular fibrillation approximately four and one-half months later. (From Detweiler, *Zentralbl. Vet.-Med.*, 4:509, 1957.)

is only 110 per minute. On the other hand, as seen in figure 5, a ventricular rate as high as 340 per minute may result with 1:1 atrioventricular conduction. As can be seen in this record it may be necessary to induce the oculo-cardiac vagal reflex, to reveal the flutter waves in such cases.

Auricular Fibrillation. In this supraventricular arrhythmia the auricles are in a state of chaotic activity and irregular waves, usually at a rate of from 500 to 1,000 per minute, replace the P waves of the electrocardiogram.[2] Impulses arising in the auricle bombard the atrioventricular node at high frequency. In untreated cases the ventricles respond with an irregular rhythm and rapid rate, usually exceeding 190 per minute. Electrocardiograms showing this arrhythmia are depicted in figures 4 and 6. The latter shows abolition of the arrhythmia with quinidine sulfate.

The occurrence of auricular fibrillation has grave prognostic significance. The duration of life after the arrhythmia was first diagnosed in the clinic varied from a few hours to 28 weeks in a series of 13 dogs.[2] Most of them died within one to three months.

Auricular and Ventricular Premature Beats. An auricular premature beat is seen in figures 4 and 7. Ventricular, extrasystoles are depicted in the latter figure. Auricular premature beats have been recorded in only three dogs in our clinic and two of these later developed auricular fibrillation. A focus in the auricle fires off independently, producing a premature beat. Whether or not they are only associated with heart disease in dogs remains to be determined. In the third case no evidence of heart disease was present.

In our experience and that of others[3,4,5] ventricular premature systoles in the dog have not been observed in normal animals and myocardial lesions have always been found in those cases which were necropsied. The cardiac impulse arises in the ventricles and is conducted throughout the ventricular musculature following a path other than that seen in supraventricular rhythms. The resultant QRST complexes are bizarre in form and usually easily recognized.

Heart Block. This term is used to designate delay or interruption in conduction of the impulse somewhere in its course from the pacemaker in the sino-atrial node to the ventricular muscle fibers. Sino-auricular block, in which the impulse fails to reach the auricles, and bundle branch block, in which the impulse is delayed or blocked in either the right or left branch of the bundle of His, are rare forms and will not be further discussed here.

Atrioventricular (AV) block indicates delay or interruption of impulse conduction between the auricles and ventricles. In incomplete AV block the conduction is merely delayed, as indicated by a prolonged PR interval which exceeds 0.15 second. In incomplete AV block with dropped beats, conduction is prevented at intervals so that the ventricles fail to contract periodically (figure 8). When conduction between the auricles and ventricles fails entirely the condition is called complete AV block (figure 9). The auricles and ventricles beat independently; the latter in response to an ectopic pacemaker in the nodal or ventricular tissue.

Incomplete AV block, with or without dropped beats, may occur following morphine sulfate medication and can be induced by other drugs which similarly elevate vagal tone (figure 8). When incomplete AV block with dropped beats or complete AV block develop owing to disease involving the conduction system serious cardiac damage is usually present. Sometimes the disturbance is transient, as in myocarditis associated with an infectious disease, and disappears when the underlying condition improves. When caused by digitalis overdosage, withdrawal of the drug will result in its disappearance.

Heart Murmurs

The first and second heart sounds are schematically depicted in figure 2 and their relationship to the electrocardiogram has been described in the foregoing. The fourth heart sound, which may be audible in cases of incomplete or complete atrioventricular block, is seen in the phonocardiogram of figure 9. When this sound is heard at the time of a periodically missed ventricular beat, incomplete atrioventricular block with missed beats can be diagnosed without the aid of an electrocardiogram. Similarly, audible auricular sounds occurring between slow, regular ventricular beats indicate complete AV block.

The most common valvular lesion in the dog is mitral insufficiency. The regurgitant flow of blood produces a systolic murmur which is most intense at the cardiac apex (figure 10). Acquired valvular lesions are most common in dogs over six years of age and the murmur of mitral insufficiency may be first detected at about that age. These dogs may live for years before showing evidence of congestive heart failure. Not infrequently dogs with mitral insufficiency survive into old age and remain compensated until their death from some other cause. In other cases the lesion may be present for some years before congestive heart failure develops. This may occur quite suddenly and death follow within a few months of the first clinical manifestations of heart failure. Sometimes the only evidence of cardiac insufficiency is pulmonary congestion and a persistent cough which may become almost continuous. Severe paroxysms of coughing may occur several times daily. In some instances such cases will respond well to digitalization and thus be made comfortable for several months or a year or two.

Triscuspid insufficiency occurs less commonly in the dog and usually is associated with valvular lesions in the left side of the heart. During systole blood passes through the incompetent valve into the right auricle, increasing the pressure in this chamber and the great veins. In severe cases the liver is enlarged and may pulsate excessively. The rise in atrial pressure during ventricular systole is transmitted as a pulse to the posterior vena cava and liver. This pulsatile expansion of the liver may be palpated through the relaxed abdominal wall (figure 11).

Reflex Vagal Slowing as a Diagnostic Aid

There are two ways which can be used to demonstrate clinically whether or not the vagus still has control over the cardiac rhythm. If it were easy to do, the most effective way would be to compress the carotid sinus. In the dog, however, this is difficult. It appears to be painful; the sinus is difficult to palpate and finally to compress. Reflex vagal excitation is induced and cardiac slowing results. A more reliable way to accomplish reflex vagal

slowing, under clinical conditions, is with eyeball pressure (the oculocardiac reflex). Figure 5 illustrates one application of the oculocardiac reflex as a diagnostic aid. In this case it was used to inhibit momentarily atrioventricular conduction and during the interval the waves of auricular flutter were revealed in the electrocardiogram. Ventricular tachycardia, *i.e.* tachycardia resulting from a rapidly discharging pacemaker in a ventricle, is not modified by elevated vagal tone while sinus tachycardia may be slowed transiently.

Contraction of the Diaphragm Synchronous with the Heart Beat

This rare condition has occurred in the clinic of the University of Pennsylvania in only four of approximately 33,000 dogs.* It is primarily of interest in the present context because of the confusion it may cause when first encountered by the clinician. These cases give the impression that there is a pulsating mass in the abdominal cavity, such as a massive aneurysm, or that the thrust of an excessively forceful heart beat is being transmitted to the abdominal cavity. Figure 12 is a record showing the relationship between the diaphragmatic spasms and the heart sounds. The strength of the diaphragmatic contraction was variable from case to case and from time to time in the same individual. This condition occurs in man associated with hypocalcemia or alkalosis, which presumably results in reduction in ionized calcium concentration and, consequently, an increased irritability of the phrenic nerves. In three of the four cases found in the dog persistent vomiting may have produced an alkalosis, although this was not established by laboratory tests.

Congestive Heart Failure

The mechanism of congestive heart failure is not completely understood, although information gained during the last two decades has contributed much toward a fuller knowledge of pertinent factors. It is apparent that the primary defect in congestive heart failure is retention of the sodium ion by the kidney. The abnormal retention of fluid is secondary to this. That sodium rather than water retention is of first importance is indicated by the observation that ingestion of sodium in animals with congestive heart failure results in increased edema fluid, whereas increased water intake rarely increases edema and may even initiate diuresis.

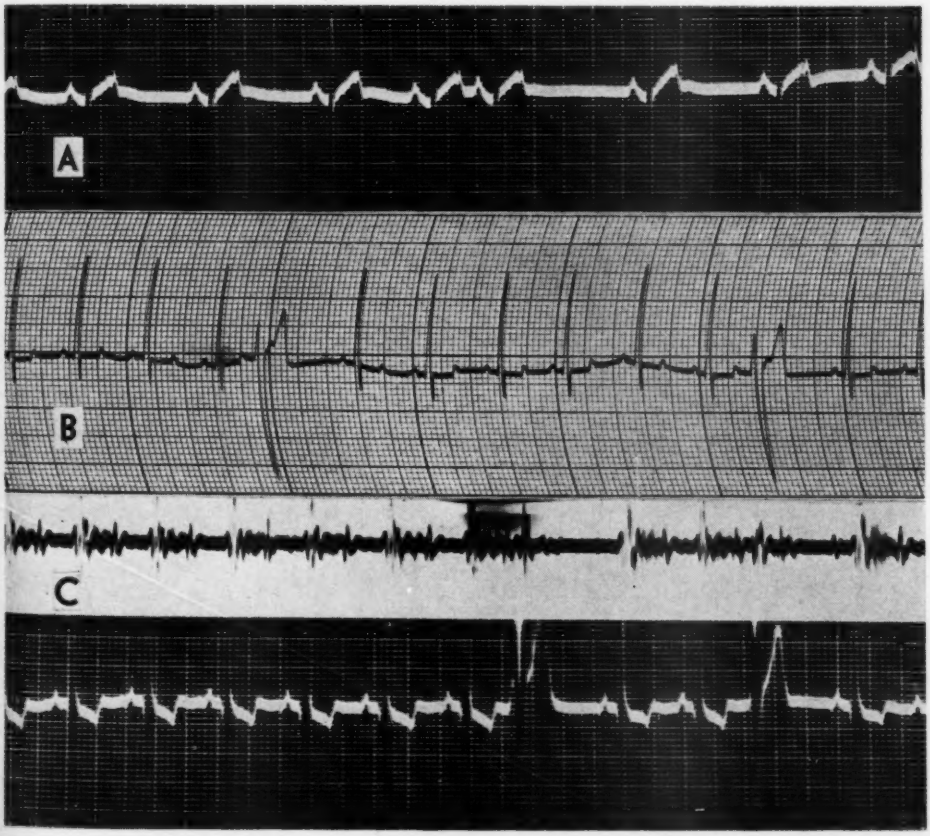
The mechanism of sodium retention must finally be sought in the renal tubule and the factors regulating its function. It will not be possible to discuss here all of the vascular and humoral mechanisms which may be involved in congestive heart failure. Attention will be restricted to known humoral influences which, on the basis of current experimental findings, appear to play a role.

It will be recalled that the renal tubule consists of a dilated capsule (Bowman's capsule) which encloses the glomerulus and leads into the proximal segment or convoluted tubule. The next section is the straight segment or loop of Henle, followed by the distal convoluted tubule or distal segment, and finally the collecting tubule through which the fluid passes on its way to the terminal collecting duct. Urine formation begins with the production of an ultrafiltrate of plasma at the glomerular membrane. This filtrate, the glomerular fluid, then passes through the renal tubule where it is modified by the processes of reabsorption and excretion. About 80% of the filtered water is reabsorbed in the proximal segment. Sodium and other ions are absorbed here also and the tonicity of the tubular fluid remains the same as that of the blood plasma. Since urine flow during maximal water diuresis is less than 20% of the glomerular filtration rate, this reabsorption of 80% of the glomerular filtrate in the proximal tubule appears to be a constant. The fixed reabsorption of water here has been designated "obligatory" reabsorption. About 20% of the glomerular filtrate remains and can be either reabsorbed or passed on distally, in accordance with the need to conserve or reject water and various solutes. The variable water reabsorption which occurs in the distal seg-

ment has been termed "facultative." Thus the final concentration of solutes in urine is a function of the distal segment. Urine volume depends on the glomerular filtration rate and the amount of the glomerular fluid volume, up to a maximum of 20%, which is not reabsorbed.

For some reason there is excessive tubular reabsorption of sodium and water in heart failure. Endocrine agents, notably adrenal cortical hormones and the antiduiretic hormone (ADH) of the posterior pituitary, appear to mediate, at least in part if not entirely, this altered tubular activity. Desoxycorticosterone and aldosterone are the most powerful sodium retaining factors formed in the adrenal cortex. Their administration causes sodium retention and may produce edema. Recently, Davis *et al.*[7] have found that aldosterone is increased in adrenal vein blood of dogs with experimental congestive heart failure, although other workers[8] failed to confirm this. Increased urinary corticords including aldosterone have been found in the urine of human patients and dogs with congestive heart failure.[7] The antidiuretic hormone (ADH) of the posterior pituitary acts on the distal tubule to increase reabsorption of water, without directly affecting sodium excretion. Among the factors which may cause release of antiduiretic substance are dehydration, increased sodium intake, injection of hypertonic solutions, and muscular exercise. Presumably decreased sodium excretion caused by adrenal mineralocorticoids results in liberation of ADH which in turn causes water retention. This hypothetical mechanism would explain the observation that in heart failure water retention is secondary to sodium retention, the untoward effect of sodium ingestion, and suggest one way through which muscular activity may increase the accumulation of

Fig. 7. A. Auricular premature contraction. B. Ventricular premature systoles. C. Phonocardiogram and electrocardiogram from a dog with aortic stenosis and myocarditis, showing a systolic murmur and ventricular premature beats. (From Detweiler in Hoskins and Lacroix, Canine Medicine, 2nd ed., in press.)



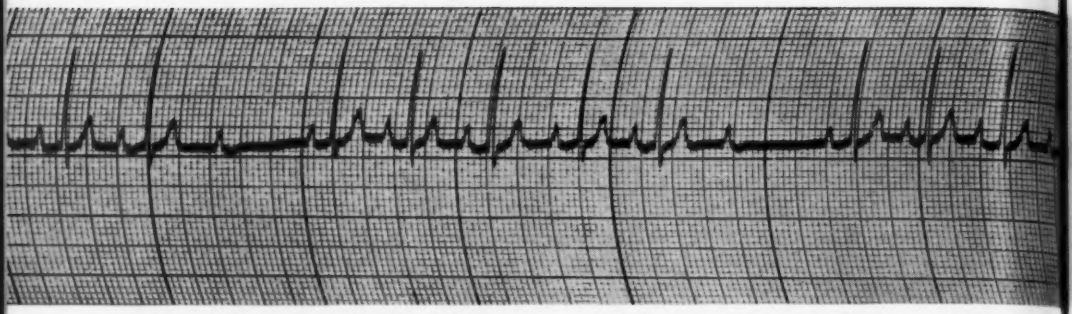


Fig. 8. Transient, incomplete heart block with dropped beats following morphine and atropine administration. Both drugs elevate vagal tone. Atropine blocks vagal action peripherally and when this effect developed the arrythmia disappeared. (From Detweiler in Hoskins and Lacroix, Canine Medicine, 2nd ed., in press.)

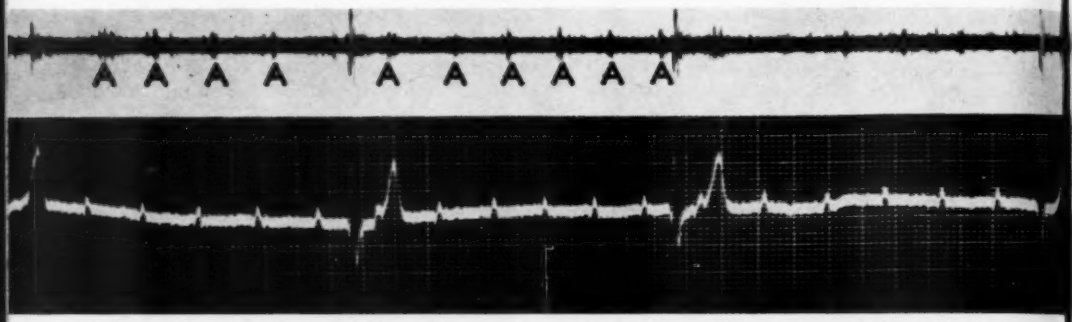


Fig. 9. Phonocardiogram and electrocardiogram from a dog with complete atrioventricular heart block. Note the audible auricular sounds labeled A. The auricular rate is 145 per minute and the ventricular rate 23 to 25 per minute. (From Detweiler in Hoskins and Lacroix, Canine Medicine, 2nd ed., in press.)

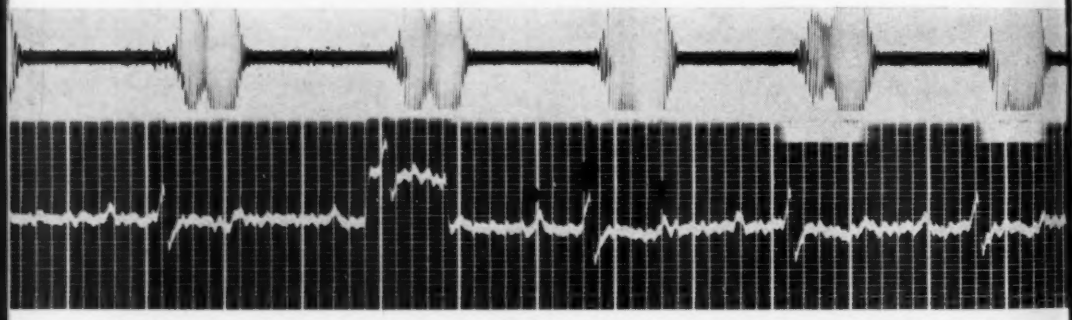
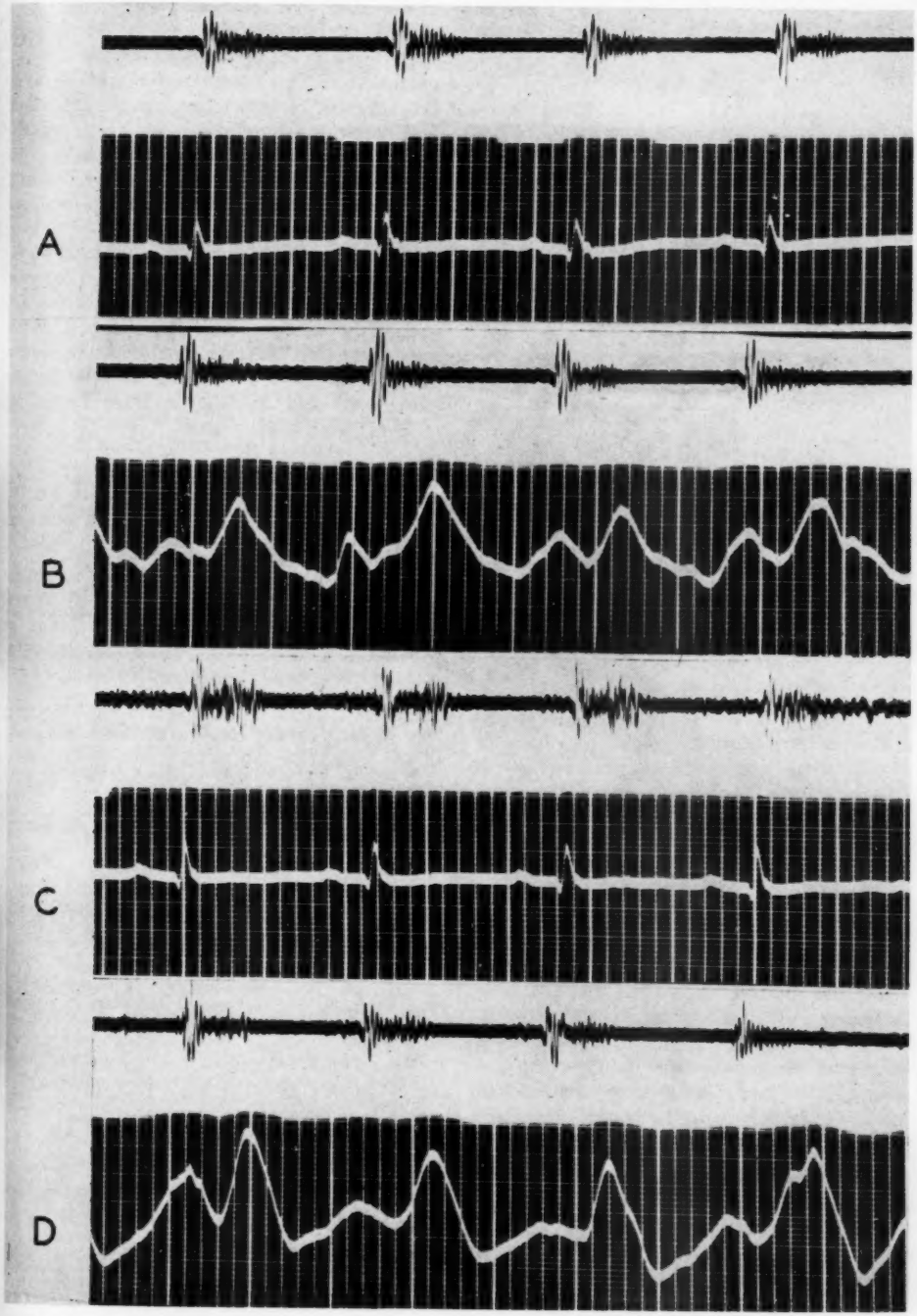


Fig. 10. Phonocardiogram (upper record) recorded over the cardiac apex and electrocardiogram (lead II) from a dog with insufficiency of the mitral valve. P R T waves are labeled in one complex. The irregular base line undulations in the electrocardiogram are caused by trembling.

Fig. 11. Phonocardiogram, electrocardiogram, and liver pulse curve from a dog with insufficiency of the mitral and tricuspid valves. A. Phonocardiogram (upper tracing) taken with the microphone at the mitral area and electrocardiogram (lower tracing). B. Phonocardiogram taken as in A. and liver pulse curve. C. Phonocardiogram taken with the microphone at the tricuspid area and electrocardiogram. D. Phonocardiogram taken as in C. and liver pulse curve. Note the marked liver pulsation occurring during the last half of ventricular systole owing to reflux of blood through the incompetent tricuspid valve and resultant elevated venous pressure and pulsation. (From Detweiler in Hoskins and Lacroix, Canine Medicine, 2nd ed., in press.)


A
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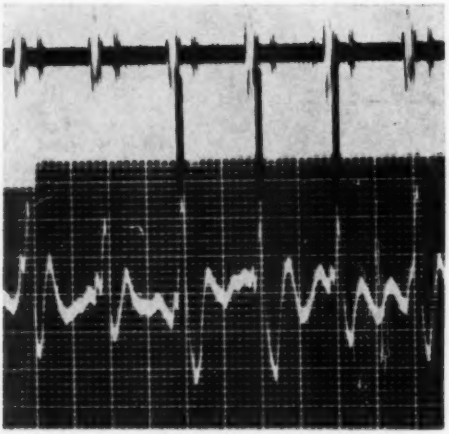

Fig. 12. Phonocardiogram (upper tracing) and record of abdominal wall pulsations (lower tracing) taken simultaneously from a dog in which the diaphragm contracted with each heartbeat. The pulse recorder was placed on the left side of the abdominal wall just caudal to the last rib. The vertical black lines extend from the peak of the deflection caused by diaphragm contractions to the first heart sound. (From Detweiler, *Jour. Am. Vet. Med. Assn.*, 126:445, 1955.)

edema fluid. These explanations remain conjectural although supported by experimental evidence.

Exactly how the abnormal renal function associated with congestive heart failure is initiated is not clear. As indicated in the foregoing, the functional activity of the nephron changes. The kidney acts to conserve water and sodium despite their presence within the body in excess. It has been postulated that this is because there is a relative reduction in cardiac output although an absolute decrease is not always observed. In any case, increased sodium and water are reabsorbed in the renal tubule and there is a resultant increase in interstitial fluid. Concomitantly, the volume of the blood is increased as indicated by engorgement of blood vessels, enlargement of the liver, and pulmonary congestion.

In treating congestive heart failure, methods are selected to support cardiac action and to modify renal tubular function. Effective digitalization overcomes cardiac decompensation. When this occurs the initiating cause is removed and renal excretion of the excess water and electrolytes results. Another therapeutic approach is to alter the function of the renal tubule with diuretics. All diuretics which are effective in congestive heart failure, inhibit reabsorption of sodium by the renal tubules. If increased aldosterone is confirmed as the essential agent which causes increased sodium reabsorption in congestive heart failure, drugs which block its action would be especially useful. Recently, it has been reported that certain synthetic steroids block the sodium-retaining action of aldosterone[9] and increase sodium excretion in congestive heart failure.[10]

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Removal of Ascarids from Pigeons

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VARIOUS PIPERAZINE COMPOUNDS have been used successfully for the removal of the large intestinal roundworm, *Ascaridia galli*, from chickens. However, these compounds have received little attention as a treatment for *Ascaridia columbae* of pigeons. Whitney[1] described a practical test in which 1,400 homing pigeons were treated with piperazine citrate at a dose rate of 8 gm. per gallon of drinking water. This treatment proved to be effective for the removal of *A. columbae*, and no harmful effects were noted.

Recently the writers tested piperazine citrate as an anthelmintic for the removal of ascarids from a small number of heavily parasitized pigeons.

Thirteen White King pigeons, which had been acquired from a pigeon fancier in the South to form the nucleus of a colony to be used for trichomoniasis studies, were found to harbor *Trichomonas gallinae* when smears and cultures from the upper digestive tract were examined. In order to prevent the transfer of these parasites from adult to offspring, these birds were treated with an aqueous solution of 2-amino-5-nitrothiazole. One milliliter of this solution, containing 10 mg. of the drug, was pipetted into the esophagus and crop of each bird every day for seven days. The treatment apparently freed the birds of the infection. Both smears and cultures were negative for several days after withdrawal of the medication. The birds were then released into a colony house.

About six months later, one of the birds died. The cause of death was not determined. Two months after the death of this bird, two others showed signs of illness. One of these was sacrificed, and many

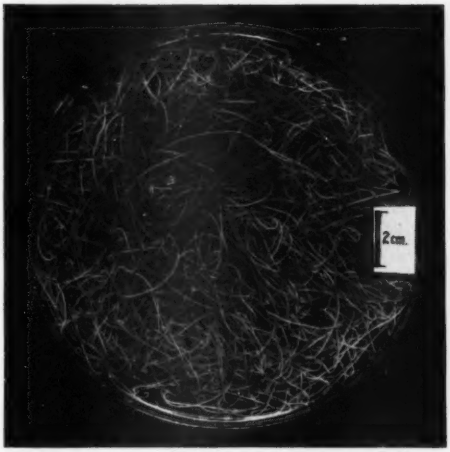

Fig. 1. *Ascaridia columbae* passed by pigeons after initial period of medication with piperazine citrate.

roundworms, *A. columbae*, were recovered from the intestine. A composite fecal sample from the other birds, examined by the brine flotation technic, contained numerous ascarid eggs.

All surviving birds were deprived of water for approximately 20 hours in preparation for treatment with piperazine citrate. After this period, an aqueous solution containing 8 gm. of piperazine citrate per gallon served as the birds' only source of water for 60 hours. Fresh solution was added to replenish that which had been consumed.

Medication removed 1,293 ascarids — 212 on the first day, 930 on the second, and 151 on the third (fig. 1). Treatment was not completely effective, however, since fecal smears taken for several day after withdrawal of medication continued to show the presence of ascarid eggs.

The second of the sick pigeons still exhibited signs of illness and was sacrificed. The intestine of this bird contained so many ascarids that the lumen was completely closed. Emaciated condition of this bird and the fact that little food was found in the intestinal tract led to the conclusion

that the bird ate and drank little during the treatment period.

For a second treatment, each bird was given 1 ml. of an aqueous solution of piperazine citrate, calibrated so that each milliliter contained 50 mg. of the piperazine base. Each day for the following ten days, droppings were collected and examined for worms. None were found. Composite fecal samples, however, continued to show ascarid ova.

A third treatment, identical to the first, was given. This resulted in the recovery of 16 additional worms. No ascarid worm eggs were seen in fecal examinations made for several days after the withdrawal of the medication. Since these birds were being held for breeding purposes, they could not be sacrificed in order to determine the effectiveness of treatment. However, it is probable that no adult worms remained; eggs were not found in repeated fecal examinations.

The results of this preliminary clinical experience indicate that a single treatment with piperazine citrate, administered in the drinking water as a continuous treatment, or given as an individual dose, is not completely effective against *A. columbae*, when used at the reported concentration.

Additional tests of a more critical nature are necessary in order to understand completely the value of piperazine citrate and other piperazine compounds as anthelmintics for the removal of the pigeon ascarid.

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Immunity to Canine Distemper

Six years ago, control measures for distemper by vaccination, especially the avianized live virus type, were practiced with high hopes. Expectations were not fulfilled. Since then, however, research programs have been initiated and more facts about distemper as a disease have been discovered and confirmed. Today, these facts again raise hopes that distemper can be conquered.

Practical consideration of the information now available has shown that past failures have an explanation, and greater success can be anticipated. This information can be classified in three general catagories: (1) Improvement of vaccine; (2) more knowledge of distemper as a disease; (3) a real understanding of dog response to immunization.

Past vaccines, while meeting potency standards, were not always adequate to immunize dogs. Recent findings which determined the amount of egg measured virus required to immunize dogs and having that amount in each and every dose of vaccine assures a perfect vaccine.

Knowledge of the pathogenesis of distemper encephalitis and the correlation between distemper and infectious canine hepatitis explained many failures. More important, this information emphasizes the need for immunization as early as possible against distemper and also against infectious canine hepatitis.

Finally there is appreciation that the dog participates in the immunization procedure which is best illustrated by the relationship between colostral protection and immunizing capacity of a dog. A puppy with colostral protection cannot be immunized or, in other words, a puppy must become completely susceptible in order to be immunized. Studies thus far have shown all puppies, whether from immune mothers or not, should be immunized when they are no younger than nine weeks of age. Of course, some puppies lose colostral protection before nine weeks of age and therefore become susceptible. Serum can be used as temporary protection but must be discontinued at least two weeks before attempting vaccination. — *James A. Baker, D.V.M., director, Veterinary Virus Research Institute, Cornell University, Ithaca, N. Y.*

Clearview
Animal Hospital

SMALL TOWN VETERINARY HOSPITAL

Clearview Animal Hospital is located at Bloomfield, Iowa, a county seat town of about 3,200. The practice is predominantly large animal, but small animal work has steadily increased during the past several years. In 1957 the hospital was built on 40 acres of land adjoining an arterial highway at the north edge of town. The site overlooks a wooded area and a pond stocked with bass and bluegill.

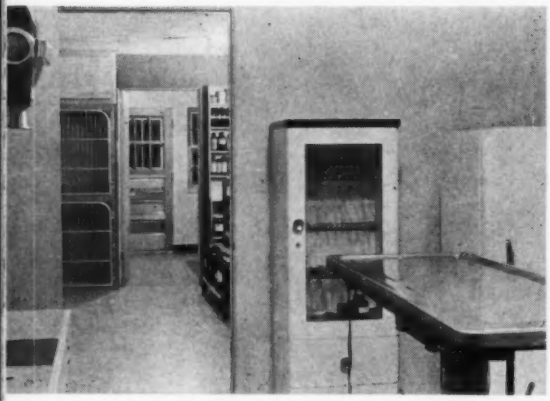
The building was designed so that it could be easily converted to a home, or remodeled for addition of wards. It is a split level, conventional type, frame, brick-faced building 56 ft. wide and approximately 26 ft. deep. The garage and carport are incorporated under the roof for all-weather access to the service entrance.

Electric heat wires in the ceiling plaster are controlled by individual room thermostats. Radiant heat allows no interchange of odors. Air movement of dust or air borne bacteria is also eliminated. Cost of this type of heat at 1.5 cents per kilowatt is not prohibitive. Electric heating wires are also buried in the sidewalk for snow and ice removal.

Walls are constructed of painted plaster and floors are covered with marbelized linoleum tile. Door and window casings are built flush without trim and all cabinet woodwork and doors are birch. Outside doors are Jalousie doors for summer ventilation and winter storm doors.

The reception room has a birch counter which encloses the receptionist's desk. The counter is faced with a knotty cedar planter filled with philodendrons and ivy. At the desk are a telephone, two-way radio and

Flowers, a knotty cedar planter and a picture window overlooking the scenic countryside, all characterize the reception room of this model hospital owned and operated by Dr. Russell E. Rasmussen, Bloomfield, Iowa.



A view of surgery, looking through pharmacy into the ward. Surgery is located on the side of the building away from traffic.

Spotless rows of bottles line the walls of the pharmacy, which is separated from the laboratory by a sink and counter. Enclosed drug cabinets are not shown here.

intercom to the doctor's home, which is one-half mile away. The picture window overlooks the landscaped hillside and pond.

In the doctor's office are the library, current professional periodicals, office supplies and a clothes closet. The room itself is furnished in knotty red cedar.

The operating room is located on the side of the building away from traffic. It is furnished with an operating and treatment table, refrigerator, small animal medicines, instruments, sterilizer, and portable *x*-ray. The darkroom and lavatory are combined.

The laboratory and pharmacy are located at each end of one room divided by a sink and counter. The two walls of the pharmacy have open drug shelves and the opposite wall has enclosed drug cabinets. One corner contains two cages for surgical patients and postoperative observation.

Stainless steel cages in the wards are sound treated with automobile undercoating on exposed surfaces. Aluminum doors open on either side, or may be removed for cleaning. One large, two medium, and five small cages are now used in the ward, and four outdoor exercise runs are located immediately outside. The removable top on the grooming table covers a deep stainless steel bathtub and utility sink.

The garage has a raised wood-floored area for bulk storage. This can be converted to a second ward. The garage proper is 14 by 20 ft. and has a large door for truck entrance, so this can be used for large animal treatment or surgery. The total garage area is 14 by 30 ft.

Stainless steel cages furnish the ward. All are sound treated with undercoating for automobiles.

A complete command of all available facts, a gift for synthesis, and a feeling for style are essential to the historian. In the story of rabies, of which this is the second part, Doctor Smithcors again proves his ability. In his book, EVOLUTION OF THE VETERINARY ART, *he served his readers more than a history of times and events. Standing on his Everest, he provided a fundamental explanation of great historical movements that gave birth to the science and art of veterinary medicine.*

The History of Some Current Problems In Animal Disease VII. Rabies

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Part 2.

Sylvatic Rabies

An early record of rabies in wildlife, and measures taken for its control, dates to an outbreak of vulpine rabies in Switzerland in 1803. Foxes entered the towns in broad daylight, biting persons and domestic animals. Postmortem examination upon a number of foxes was ordered by the Sanitary Commission, and a general hunt was ordered, with the result that the disease disappeared from Switzerland, but not before it had spread to neighboring countries. An official report from Wurtemberg, where the disease had been allowed to exist for some two decades, is of interest in that it states, concerning persons and animals bitten by foxes but who did not develop the disease: "These exemptions lead those connected with hunting pursuits into the fallacious reasoning that the malady was not contagious." The government commission, however, appears not to have been swayed by those who had a selfish interest in preventing the general destruction of this wildlife reservoir.

Numerous outbreaks of rabies in wildlife are on record in Europe and America, and the Europeans seem to have had little compunction in ordering wholesale destruction of the offending species once the nature of the infection was established. The damage that can be done by one such animal is demonstrated by the fact that one rabid wolf in France in 1851 bit no less than 46 persons and 82 head of cattle in one day; the cattle were all destroyed, and many of the bitten persons died. And in Turkey in 1852, one wolf bit 128 persons and 85 sheep.

In 1856, rabies was communicated by dogs to a herd of deer in England, of which more than a hundred died with symptoms of the disease, whereupon, as reported in the *Veterinarian* (1857), the medical officer of the local board of health "issued a timely notice or caution to the inhabitants . . . to be on their guard to protect themselves and their families from the infliction of this terrible malady." It may be doubted, however, that a "timely warning" would constitute an effective control program, although this at least would be an improvement upon some of the ancient ritualistic measures designed as preventives. A number of these "control programs" survived until relatively recent times, and undoubtedly are still practiced in some areas. For example, during the late 19th century, when rabies was rampant in Ethiopia, dogs were regularly gathered into huts filled with straw; the straw was ignited and the dogs forced to pass through the flames to escape. This was considered a "certain preventive" of rabies. If we are amused by such primitive customs, it might be noted that the last "fire drive" — to protect animals from disease — occurred in Scotland in 1826, and in Germany in 1828. And a note in the *Veterinarian* for 1853 suggests the origin of a *bon mot*, used today under perhaps more innocuous circumstances. It appears that it was the custom of Scottish shepherds to apply a few hairs pulled from an offending dog to bite wounds in the belief that "the hair of the dog that bit you" had "virtues either as a curative or preventive agent" against rabies.

Effective Control

A major step toward effective control was taken in England following an outbreak of rabies of "formidable extension and virulency" in 1866. In 1867 a "Metropolitan Streets Act" was passed which enabled the police to seize all stray dogs. When the act was put in force in 1868, the number of rabies deaths among persons was greatly diminished. But human nature being what it is, enforcement became lax, and in 1872 another great outbreak occurred, of which Fleming wrote: "The wide and serious extension of this epizooty over the country appears to have been largely, if not altogether, due to the insufficiency of the police measures adopted in the different towns and districts, the late period at which they were introduced, the want of a proper and uniform sanitary organization to combat the spread of this and other contagious diseases of animals, and the general ignorance prevailing with regard to its symptoms and nature."

With respect to rabies, the existence of this disease in significant proportions in any country can be traced to a failure to recognize the importance of any one or more of these factors itemized by Fleming. Having learned her lesson by bitter experience, England today, and for some years past has been rabies-free (since 1921, and except for one infected dog "smuggled" into the country in 1918, would have been rabies-free since 1903). Unfortunately, the same can not be said for the United States. Without detracting from the enviable position of Britain today, however, the fact that effective control programs had been outlined a century before they were put into effect will bear some consideration.

Capt. Bush has recently (*Jour. Am. Vet. Med. Assn.*, [Aug. 1], 1957) brought to light an informative and valuable addition to the early knowledge of rabies by presenting an article by a surgeon, Francis Hopkinson, entitled Facts and Observations Relative to the Rabies Canina, which appeared in the *Sporting Magazine* (London) in 1793. In keeping with the tenor of the times, this surgeon could not divest himself of the possibility of spontaneous generation of the disease; but excluding this as a major factor, he makes two statements worth repeating:

1. "We know of no cure when the disease has taken place; that our preventatives are very doubtful indeed; that in the human species, an early and proper attention to the wound is the only thing to be depended upon, and that the degree of danger is to be estimated from the extent and depth of the wound."

2. "In dogs, a long and continued and separate confinement is the only thing to be relied upon."

Hopkinson recommended confinement for

COUNTENANCE OF A HORSE WITH RABIES.

Line drawing plate from Manning's Illustrated Stock Doctor, Philadelphia, 1880.

12 months; Britain today requires a quarantine of six months for dogs entering the country.

Spontaneous Hydrophobia

An even more important chapter in the forgotten history of rabies is an obscure section on "Canine and Spontaneous Hydrophobia" in a volume of *Medical Reports* (1807) by the British physician, Samuel Bardsley. The report on hydrophobia first appeared in a literary review in 1793 — and thus is of the same date as Hopkinson's article — and was occasioned by a case of human rabies which occurred 12 years after the bite of a dog supposed to have been mad. Bardsley's work has received little notice in medical histories, and when it has, he has been more than once misquoted on the matter of "spontaneous" hydrophobia. By this term Bardsley meant rabies occurring in the absence of a known bite.

The student of psychosomatic medicine would delight in Bardsley's explanation of this supposed case of rabies occuring 12 years after a bite by a supposedly rabid dog. The symptoms with which the man died "leave no room for doubt concerning the resemblance of the disease to that which is the offspring of the canine poison." While he admits the possibility of the rabies virus lying dormant for as long as 20 months, with devastating logic Bardsley draws upon a host of cases with intervals up to 40 years, and demonstrates that these were most likely cases of hysteria with "the anomalous symptom of an inability to swallow fluids," or diseases bearing some resemblance to rabies, *e.g.*, tetanus. He further demonstrates that "Hydrophobia [as a symptom rather than the disease], with all the pathognomonic symptoms of Rabies Canina, has been produced by other occasional causes than the infection of a rabid animal."

In the case mentioned, symptoms began with inability to swallow, and "being impressed with a remembrance of his having been bitten [12 years previous] by a supposed mad dog . . . the image of the dog haunted his imagination with perpetual terrors; and the expectation of a violent death, by being smothered—a vulgar and unjust persuasion too often entertained — would not a little tend to increase the nervous irritation already excited." The fear of being smothered refers to a provincial custom best explained in Daniel's *Rural Sports* (London, 1813), in which the author states: "There is a vulgar Prejudice, that a Person bitten by a Mad-dog, and pronounced irrecoverable, may, according to the LAWS OF THE LAND, be bled to Death, or smothered. To correct this Prejudice, We quote Sir Vicary Gibbs, 1807, on this Point.

" 'I am clearly of Opinion, that it is not lawful, by any Means, wilfully to put to Death, a Person who has been bitten by a Mad-dog; and those who commit such an Act, are guilty of MURDER.' " Daniel records one instance of a child with rabies being smothered by the parents, who were tried for murder and convicted, but later pardoned.

Circumstances such as these, and not spontaneous generation of the virus of rabies, is what Bardsley meant by "spontaneous hydrophobia." Bardsley says: "I wish it to be

understood in a sense different from that, in which it is commonly used . . . I do not conceive, in such instances, any specific poison to have been generated in the habit." He further asserts that he has acceded to popular custom in using the term at all, for "the assumption of such a title as spontaneous Hydrophobia is calculated to divert the attention from sober enquiry, and to involve a subject sufficiently obscure in still greater darkness."

With equally persuasive logic, Bardsley disposes of the more commonly held opinions on the origin of rabies in the dog. Concerning the consumption of putrid flesh, he mentions the work of James Mease, port physician of Philadelphia, who in 1792 published *An Essay on the Bite of a Mad Dog*. Like Bardsley, Mease contended that rabies arose only by the innoculation occasioned by the bite of an infected animal, but held that in the great outbreak in Philadelphia in the 1780's a contributing cause was the huge mass of dead animals that was allowed to putrify on the commons at the conclusion of the late war. Bardsley quite correctly notes that the mere congregation of dogs at the site was the exciting factor: "If only one dog were in a state capable of communicating the disease, what havoc must he make among such a multitude!" And further, "When this unrestrained assemblage of dogs was broken up, canine madness became less prevalent." In similar manner, Bardsley disposes of the supposed effects of climate, deficiency of water, and want of perspiration. On the matter of the "worm under the tongue," Bardsley states that "the testimony of Dr. James — who was a celebrated dog-doctor — is decisive upon this point: 'Dogs thus treated, run mad equally with those who have never suffered this absurd operation.' "

Bardsley continues: "Having, I trust, clearly demonstrated the improbability of canine virus being generated by the operation of any of the remote causes just enumerated, I shall now endeavor to establish the contrary opinion — *viz.* That the disease is always produced by an actual communication of the poison of an infected animal." Concerning cases of so-called spontaneous rabies, he holds that if sufficient inquiry were made, we would "discover some strange dog affected with this complaint communicating it as he passed through towns or villages . . . Why then unnecessarily multiply causes when one is sufficient to explain the effect?" In this respect, says Bardsley, rabies has an analogy to syphilis in that it is sometimes an easier course to blame it upon the atmosphere than to trace the real cause. "They who by their rank, sex, and station, were more particularly exposed to disgrace from a detection of the origin of their malady, gladly availed themselves of so convenient a doctrine." Both diseases, he contends, are "capable of being checked by due regulations."

"Preventive Scheme"

On the means for eradication of rabies, Bardsley states: "The opinion I entertained so far back as 1793 of the purely contagious nature of the disease in dogs, and the great probability of its entire extirpation by suitable regulations, is founded upon a fair induction from facts . . . The plan is as simple as I trust it will prove efficacious. — It consists merely in establishing an universal quarantine for dogs within the kingdom, and a total prohibition of the importation of these animals during the existence of such quarantine. The efficiency of this preventive scheme rests upon the validity of the following propositions.

"1st. That the disease always originates in the Canine species.

"2nd. That it never arises even in them spontaneously.

"3rd. That the contagion, when received by them, never remains latent more than a few months.

"If these propositions have been established, it clearly follows, that by destroying every dog in which the disease should break out during strict quarantine, the propagation of the malady would not only be prevented, but the absolute source of the poison entirely cut off . . . It would undoubtedly be erring on the safe side, if the time of quarantine should be extended to the very unusual period of eight months . . . I am indeed persuaded that two months would justly answer this purpose."

Bardsley's first proposition, of course, is open to question, but it should be noted that he appears to be dealing with the ultimate origin of the disease in antiquity, the controversy respecting which, he says, "would lead me into a labyrinth." His program, however, does not take into consideration wildlife reservoirs.

On the means for putting his program into effect, Bardsley notes that "It is not always the certain knowledge of an evil that leads to due exertions for its removal. We often timidly sink under ills which are supposed to be irremediable . . . What remains then to be done? Preventive means ought certainly be adopted. . . . Our insular situation is peculiarly favourable for the experiment; and the present period most propitious for the attempt . . . some one quarter or other of the united kingdoms is scarcely ever free from the presence of this terrible malady. A ready acquiescence and co-operation on the part of the people may therefore be expected, with any measures of prevention which an enlightened government should enact . . . The evil is great, has been encreasing, and can only be diminished by legislative interference."

Delabere Blaine

That "legislative interference" did not come about as a result of Bardsley's urging is evident from the rabies outbreaks of still greater virulence which occurred from 1806 to 1820. The eminent veterinarian — formerly a human surgeon — Delabere Blaine, in his work on *Canine Pathology* (1817), states that he had attended as many as five cases a day, and not infrequently the owner as well as the dog, for weeks on end. Together with his partner, the illustrious William Youatt, Blaine undoubtedly saw more cases of rabies in dogs than any other veterinarian of the times. In addition he relates that he personally attended over 50 bitten persons, a number of whom died. The section of his book dealing with rabies occupies over 60 pages in the third edition (1832), and should be read by everyone concerned with the problem today. While he deals more with the strictly medical aspects of rabies than with its public health implications, his views are refreshingly "modern," and actually earned him the enmity of the majority of medical men and veterinarians of his time, primarily because he championed the specific nature of the rabies virus.

Blaine says: "There are authorities of respectability who maintain that rabies is yet spontaneously generated; and if such be the case, no legal enactments of whatever severity, short of extinction of the whole race, could secure us . . . My own opinion, formed on an acquaintance with it of twenty-five years, in a great many of which the rabid cases amounted to several hundreds per year, is most decidedly in favour of the contagious origin of the disease, and none other." And his former partner Youatt, whom Blaine concedes had by this time seen even more cases, held the positive opinion that "rabies is produced by innoculation alone."

Blaine clears up several of the doubtful issues raised by Bardsley, and refutes a number of his errors. Bardsley's failure to consider the possibility of a wildlife reservoir of rabies may be explained by Blaine's statement that vulpine rabies was "extremely rare," and, of course, wolves were not the problem in England that they were upon the continent. Bardsley admitted the communicability of rabies to the several domestic and a number of wild species, but thought that only the Canidae could transmit it. Blaine included all quadrupeds as susceptible, and "the virus of every rabid animal will communicate the disease." On the matter of passage of the virus through the skin, Blaine refuted Bardsley's claim that it could pass through an unbroken mucous membrane, and contended that innoculation could occur only on an abraded surface — otherwise he would have long since died of the disease. He states that the incubation period in the dog ranges from one (rarely) or two weeks to two or possibly four months.

Blaine was disturbed that "thousands of innocent dogs have been sacrificed to mistaking this disease for some other . . . and on the other hand, not unfrequently dogs really rabid have been fondled, and had remedies administered to them, at great

personal risk, from a supposition that they laboured under some other complaint." He mentions epilepsy, tetanus, spasmodic colic and distemper as the most common sources for error. With regard to the experiments of Edward Jenner about 1800 in an attempt to find a vaccine for distemper (following his epochal work on smallpox), Blaine not only suggests that Jenner was dealing with rabies, but makes the serious charge that he full well knew this to be the case. This may be an instance of the even-tempered Blaine allowing personal prejudice to sway his better judgement, for he was a detractor of vaccination.

Blaine readily admits the hopelessness of effecting a cure in either man or beast once symptoms of rabies have appeared, but he extolls the virtues of a number of drugs, notably boxwood, in preventing the disease following a bite. However, he urged prompt cautery or excision of the bitten part as the surest prophylaxis. In recommending medical prophylaxis, Blaine admits he was on the horns of a dilemma. "As justly observed by Mr. Youatt, the imputation of quackery is very generally associated with the recommendation of these preventives. But . . . are there really no antidotes? . . . Let the chances be ever so small of a prophylactic property in any internal remedy, they should be cherished and employed; and foul befal him who, for personal gain, would keep the knowledge of any such matter a secret. He, indeed, would be a quack; let the profession hunt him down without regret." With some logic, Blaine asks what other course is left in cases with lacerations so extensive as to preclude effective cautery, or where excision is an impossibility.

In Watson's *Practice of Physic* (1845), one of the better medical texts of this period, the subject of rabies is handled well — in part because of the dependence placed by the author upon the judgement of Youatt. On causation, Watson refutes those of his profession who would believe either that the disease did not exist, or was spontaneously generated, or that the disease in man was susceptible to treatment. His "vest pocket" description of the course of human rabies is a gem: "A man is bitten by a dog. After a time the symptoms proper to hydrophobia come on. After another interval the man is dead." Regarding the use of drugs as preventive treatment after a bite, he says, "I have no faith in these preventives. But sometimes some of them must be tried; and I would prefer those which are sanctioned by Mr. Youatt's good opinion to any others." If he were affected with rabies himself, he says he would take opiates, "not so much in the hope of recovering as with a view to euthanasia."

Youatt's Writings

Youatt's thoughts on the matter are stated in the *Veterinarian* for 1838, in which he, like Blaine, asks: "Are there any preventives? I know that I am treading here on dangerous ground. There are plenty of these preventives, if the advertisements of the quack or the firm belief of the peasant can be depended upon. Indeed, the principles and the practice of legitimate science acknowledge the possibility of their existence . . . I have no manner of doubt that the successful treatment of rabies will ere long be discovered. This will, indeed, be one of the noblest triumphs of medicine. It is one that seems especially allotted to the veterinary surgeon. He sees the disease in all its various forms in the animal that is most frequently the subject of it."

Not only did Youatt have a most extensive experience with rabies, he also wrote copiously and well upon the subject — some 125 pages in the *Veterinarian* for 1837 and 1838 alone, and the index to the first five volumes of this journal, of which he was editor, contains two full pages of references to the subject. Like his partner Blaine, Youatt wrote with much discernment on rabies, and based his opinions on "plain, broad, palpable facts." It may be doubted that any pertinent fact concerning the medical aspects of rabies, including its epidemiology, that could have been determined at the time escaped him, and he was free to admit the existence of gaps in knowledge. Thus he says, "Of the nature of the virus we are totally ignorant. It has never been analyzed." But, unlike many of his contempor-

aries, he recognized that the virus was found only in the saliva of an infected animal, and could be communicated only through a break in the skin.

In the absence of "one decisive proof to the contrary", Youatt suggests: "It would appear that, if a species of quarantine could be established, and every dog were confined separately for eight months, the disease would be annihilated in our country; or could only reappear in consequence of the importation of some infected animal." This is precisely what happened in England in 1918 after being rabies-free 15 years. He had doubts, however, that such a restriction could be enforced, "either in the sporting world, or among the peasantry." Youatt apparently knew whereof he spoke, for he states that pitfighting, which had been outlawed, still was carried on extensively as a *sub rosa* practice, and not only did these dogs serve as a covert reservoir of rabies, but were given every opportunity to transmit the disease. He charges that "In almost every large town a dog-pit, a perfect nursery of crime of every description, is to be found . . . Without the slightest hesitation, I will affirm, that rabies is propagated, nineteen times out of twenty, by the cur and the lurcher in the country, and the fighting-dog in town." Youatt suggests a tax on dogs, and elimination of those unlicensed, as the most effective means of controlling the dog population.

The French System

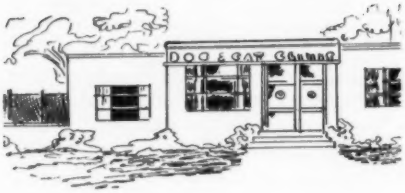
The ever fair-minded Youatt, in his capacity as editor of the *Veterinarian*, inserted along with his own articles on rabies in 1838, a paper by a French veterinarian which contained much which was at variance with his own beliefs. The operation of the French system of sanitary police in regard to rabies is of particular interest. Owners of an animal which became rabid were required to report the occurrence to the mayor or the police commissioner, who, assisted by a veterinarian, was to adopt "such measures as may be deemed expedient." On the declaration of the veterinarian that the animal was rabid, it was to be destroyed, or if doubts existed, it was to be confined. Wandering dogs were to be caught and confined, or killed if necessary. It is of considerable interest that in all cases the civil authorities were to be guided by the veterinarian. Confinement of not less than 16 days, followed by muzzling for two months if the dog was allowed to go at large, was required, and in all doubtful cases, confinement, rather than destruction, was urged. Similar measures were applied to other species. In 1821 a French physician was sentenced to pay 8,000 francs (then a considerable sum) to the widow of a man who died of rabies after being bitten by the physician's dog. The physician had been aware of the injury, but had failed to confine the dog.

In an inquiry on rabies conducted by a committee of the House of Commons in 1830, it is evident from the testimony of a number of medical men that most of them approved of the tax which had been imposed upon dogs, and that by lessening their numbers, it had an effect in reducing the cases of human rabies. One suggested muzzling, as was done in Italy, where poisoned meat was put out to take care of those not properly muzzled. But another, as spokesman for the majority convinced of the spontaneous generation of the disease, felt that "we are not yet in possession of any preventive . . . I know of no security beyond that of keeping out of the reach of accident."

On the matter of muzzling, which was required in some English municipalities in the 1850's, a number of persons had registered objections to the law — even as now — on the grounds that rabies was then "a disease exceedingly rare." In agreement, William Percivall, editor of the *Veterinarian* (1852), replied, "Thank goodness and the dog tax! so it is." James Law, in his *Textbook of Veterinary Medicine* (1900), states: "Where the muzzling of all dogs has been rigidly enforced, as in many German cities and districts, the disease has been practically extirpated . . . Berlin in 1853 had many cases of rabies and muzzling was enforced. In three years the disease was completely eradicated and the city enjoyed nine years of immunity or so long as the law was enforced."

To be concluded in a subsequent issue.

CLINIC



CLIFFORD L. McGINNIS, D.V.M.
Peoria, Illinois

Worm Conscious

I was sitting in my office and thinking of things I like when a woman entered. She had a purposeful gait, and a black Cocker in tow. She pointed accusingly at the dog and said, "He licks himself and eats rich, Doc, so he must have worms." The patient was as good an example of a healthy dog as I ever hope to see. But many owners are worm conscious; probably a result of the boiled down reading material available these days.

Anal Glands

A colleague asked what to do for the dermatitis we see on older dogs involving buttocks, perineum, or inner aspects of the hind legs and the hocks. The skin thickens, the area is denuded and it may or may not be moist. This is primarily a fungus infection, but often anal glands are infected. Surgical removal of the anal glands is a tedious operation to be avoided when possible.

A few years ago my associate, Doctor England, started to inject infected anal glands, using a mastitis tube of antibiotic ointment and it has healed most of them. The procedure is as follows: First, squeeze out the contents of the gland. By holding the tail firmly upward and forward, both ducts are exposed. Insert the tip of the ointment tube and inject both glands until the ointment oozes out past the nozzle. With a little patience and practice, this operation soon becomes a routine matter. We prefer an ointment base that is soft but not liquid, rather than the creamy liquid type of mastitis preparation. Clients seem to prefer to return their pets three or four times for treatment, rather than submit them to an operation.

American Neighbors

Legends born many centuries ago have it that Mohammed's coffin ascended into space and could be seen crossing the sky from time to time. This was before the day and age of Sputniks. It has sometimes occurred to me that "space" might make a wonderful burying ground should the land area available for such purposes suddenly become too expensive. In any case, some friends or neighbors will have to stay behind to accomplish the necessary mechanical manipulations. It has me worried.

When I was a youngster living on the farm, it was customary for neighbors to take turn "sitting up with the deceased." Neighbors not on duty would bring food and coffee to the wake so that the selected two or three sitting up would have sufficient to stay awake. The body was usually prepared at the funeral parlor and returned home to lie in state.

Either way, it worries me that my wife might have difficulty getting some one to sit up with me. I presume I have enough friends, but I haven't enough neighbors. The more I think about it, the more appalled I am by the fact that I do not know any of my neighbors well enough to ask them to sit up with me if I should die, or to trigger me into space. That is a sad commentary on the American way of life.

·VM·

If farmers delayed spring work the way politicians delay legislation for maximum of political mileage, a lot of people would be more hungry than disgusted.

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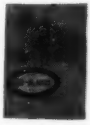

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Clinical Use of Injectable Neomycin and Polymyxin B

W. L. BAKER, JR., B.S., D.V.M.

Kennett, Missouri

OVER THE LAST YEAR, the opportunity has been presented to evaluate clinically a new synergistic combination of neomycin sulfate and polymyxin B sulfate in various systemic infections that occur routinely in a mixed veterinary practice.

The problem of increased resistance of organisms to penicillin, streptomycin, broad spectrum antibiotics, and sulfonamides has been a serious one in my practice. This may be due to promiscious use of the products by owners in improper dosage that allows development of resistant pathogenic strains. Low level feeding of antibiotics may also contribute to resistance observed in some cases. When antibiotic sensitivity disc culture study or history of the case indicate the use of broad spectrum antibiotics, we frequently face another difficulty. Continued dosage may be necessary to control infection, and these drugs are not always well tolerated by the surrounding tissue following repeated injections, even though material is properly diluted, careful technic is employed, and injection site carefully selected. These problems have not been observed with combination of neomycin and polymixin B possibly because the formulation is not used promiscuously. This combination consists of neomycin sulfate 100 mg. and polymyxin B sulfate 100,000 units per cc. in an aqueous solution made available as daribiotic® injectable which will remain stable at room temperature for at least two years.

Indications and Dosage

After six years experience in using neomycin in the laboratory and after treating approximately 300 patients Waisbren[1] stated, "It has the widest antibacterial spectrum of any of the antibiotics. It is the only antibiotic with a wide antimicrobial spectrum that is bactericidal; the concentration that will inhibit bacteria will usually kill them."

Neomycin is effective against Gram-negative and Gram-positive organisms that are found commonly in animal infections. It has a wider range of antibacterial activity than bacitracin, penicillin, or streptomycin.

Polymyxin B is also bactericidal rather than bacteriostatic, and when combined with neomycin exhibits a synergistic action *in vitro*.[2] This synergism increases the action and decreases the amount of each drug needed to control organisms involved. Polymyxin B is bactericidal to numerous Gram-negative organisms. It is particularly effective in treating infections caused by or complicated with Pseudomonas organisms. This combination of antibiotics does not produce readily resistant strains of bacteria.

After a single intramuscular injection of 1 cc. of the combination per 100 lb. of body weight in cattle, a therapeutic blood level is reached within 30 minutes which last for at least 12 hours.[3] Quite often two or three injections has been sufficient to control infections caused by susceptible organisms. In large animals, a single large dose that gives high immediate blood level will destroy susceptible organisms present and control infection. This is particularly useful in controlling septicemia as a herd infection. After approximately one year's experience in using this combination, covering several hundred cases, no toxic symptoms has been noted. It is active against infections caused by Streptococcus, Staphylococcus, *Escherichia coli, Aerobacter aerogenes, Klebsiella pneumoniae,* Vibrio, Proteus, Pseudomonas, Salmonella, Hemophilus, and Pasteurella. The dosage used and recommended for large animals is 1 to 2 cc. for each 100 lb. of

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body weight initially, and repeated in 12 hours. For small animals dosage is 0.5 to 1 cc. depending upon the size of the patient and is repeated in 12 hours.

Clinical Trials

Patients treated were classified into three categories:

(1) Those that had not responded to other antibiotics, (2) those suffering such diseases as distemper, et cetera, where previous treatments had proved unsatisfactory, and (3) where time and the situation allowed a sensitivity interpretation pattern which was computed for the organism and neomycin-polymyxin B used when so indicated.

Results and treatments are given in table 1.

TABLE 1.

Condition	Cases	Animal	Recovered
Shipping Fever	68	Cattle	60
Septicemia actinobacillus	1	Cattle	1
Klebsiella	4	Cattle	4
Escherichia coli	10	Dogs	10
Tetanus	3	Horses	3
Salmonella	16	Swine	15
Distemper complex	28	Dogs	20
Streptococcic septicemia	8	Swine	8
Foot rot	9	Cattle	9
Calf diphtheria	6	Cattle	6
Strangles	6	Horses	6
Tonsilitis	14	Dogs	14

Summary

Advantages of the relatively new injectable antibiotic combination is discussed. Its value in treating susceptible infections both in large and small animals is tabulated. The dosage recommended for large animals is 1 to 2 cc. per 100 lb. of body weight, and for small animals 0.5 to 1 cc. per animal repeated every 12 hours as indicated.

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Stress and the Host-Parasite . . .

Continued from page 241

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Distaff

MRS. E. F. (HAZEL) EBERT

Wife in the Doctor's Office

I have always counted it a curious experience to be the wife of a practitioner. In many cases I have taken the role of secretary, bookkeeper, receptionist, charwoman, part-time help and full-time standby for anything required around a veterinarian's office. Looking back, I suppose that I have sometimes responded like a watered-down version of Doctor Jekyll and Mr. Hyde. One can not easily adjust to the extremes of that grand diversity that invades a practitioner's office. Yet there are a few basic rules that apply to most people and situations.

First of all, be yourself. Embalm any artificial airs you may have acquired, and lay them to rest for good. Pomp without power is only ridiculous. Remember, the power is in the examining room and surgery, not behind the receptionist's desk. Simply be friendly and interested.

Receiving the Client

In a large city the lack of "personal interest" between clients and practitioners may be felt, but it is a situation that can be remedied. I have found that city folks love their animals just as much as do people from the country. A veterinarian's wife can make a career of meeting her husband's clients and creating some of the personal interest atmosphere frequently lost in the tempo of modern day living. Of course it is much easier if she is sincerely interested, for there can be no better way to establish the bond of friendship.

Not only does the veterinarian-client personal interest created by the distaff side of the practice pay off in dollars — I know from experience that it is rewarding by way of friendship and kindness. For every hour that I have spent in the office I have been paid many times by the thoughtfulness of my husband's clients.

There may be a few undesirable clients in any practice, chiefly those who do not understand nor appreciate what the veterinarian is doing for them. But most clients who come into the office or call by telephone are unaffectedly genuine. It is a pleasure to give them your time and interest.

The Case History

One of the many functions a wife can perform for her husband in certain types of practice is case history taking. Clients who avail themselves of veterinary service run the gamut of problems. An astute wife-receptionist can tactfully prevent many people from squandering her husband's time. An adequate case record card takes in as much history as possible, and sets down complaints, real and imaginary, and some of their causes in a codified form. Perhaps not all the case history can be taken by the wife-receptionist, but she can make a good start on it. Tact and patience are all important. It takes patience to elicit information from a client one syllable at a time, and patience comes with practice.

Tact, like "cause," is one of those words we all think we understand. I used to tell the story about Mrs. Jones and her bridge game. If one of the ladies mentioned a dog sick with pneumonia, within three days three new cases of pneumonia materialized at the hospital — by odd coincidence all of them belonging to the bridge players. When Mrs. Jones had two tables of bridge, business would boom with seven new patients, all "exhibiting" similar symptoms. The power of suggestion can replace a "cause," but nothing can replace tact.

"Welcome"

A distaff was an important bit of equipment for our great grandmothers, but it was no more important than the welcome at the veterinarian's office or clinic. Welcome means little unless we express it when clients or visitors enter the office or home of the veterinarian we represent.

Practitioner's Laboratory

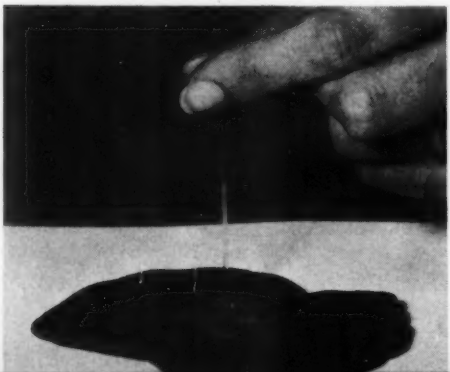
W. L. SIPPEL, V.M.D., Ph.D.
Florida Livestock Board
Kissimmee, Florida

The Hematocrit Test

Figure 1.

Hematocrit centrifuge

Fig. 2. Sealing capillary tube with modeling clay.



The hematocrit test is one of the more valuable clinical laboratory tests available to the practicing veterinarian. Probably more information can be obtained through its use than through the use of any other test. This simple technic provides the following information:

1. Packed cell volume (hematocrit).
2. Sedimentation rate.
3. Buffy coat.
4. Icterus index.

Recently a relatively inexpensive high speed centrifuge for hematocrit determination (fig. 1) has become commercially available.[1] This instrument has two great advantages for practitioners: Only a drop of blood is needed to fill the capillary tubes used and only four minutes are required to furnish the packed red cell volume reading. Dr. C. E. Bild of Miami, a prominent laboratory-minded practitioner, has stated that his three most useful instruments are his microscope, his sterilizer, and his hematocrit centrifuge. Information of a nonspecific nature gained from this test is fully as valuable as that derived from the clinical thermometer and total or differential white blood cell counts. It should be part of the routine examination of all patients in the hospital and elsewhere if practicable.

This article will deal with the hematocrit test, and be followed in a subsequent issue by the others listed above.

Technic — Hematocrit Test

Two methods are available, the capillary tube and Wintrobe tube technics. The capillary tubes (fig. 2) contain heparin as an anticoagulant and are filled by capillary attraction from a drop or two of blood. The conventional Wintrobe tubes (fig. 3) are larger and are usually filled with venous blood to which an anticoagulant has been added. (For 5 ml. of blood use 0.15 ml. of a solution containing 4 gm. of potassium oxalate and 6 gm. ammonium oxalate in 100 ml. water, dried on the side of the tube with gentle heat.)

Wintrobe tubes can be conveniently filled with a 4½ in. 20-gauge needle and syringe. Drawn out glass tubing for the purpose

breaks too easily. Capillary tubes without heparin are available and can be used with the above anticoagulant.

Bild[2] has suggested filling two heparinized capillary tubes with the patient's blood. One tube is immediately placed in the centrifuge for the hematocrit reading. The other is used for a preliminary sedimentation test to determine the desirability of a more accurate test with other equipment.

After filling with blood treated with an anticoagulant, the hematocrit or sedimentation tube is placed in the centrifuge. The capillary tubes are made of soft glass and are open at both ends. Before centrifuging, one end must be sealed in a laboratory burner or plugged with modeling clay. The new high speed hematocrit centrifuges require four minutes of centrifugation. The conventional Wintrobe tube method requires spinning for 30 minutes in a centrifuge that will develop at least 3,000 r.p.m.

After centrifugation for the required time, the height of the packed red blood cell column in the bottom of the tube can be read directly in percent from the Wintrobe tube. With the capillary tube, the packed cell volume must be calculated by dividing the total height of the blood column in millimeters into the height of the packed red cells. The answer, multiplied by 100, is the percent hematocrit reading. Instruments which make these calculations automatically are available.

Wirth[3] quotes Lederhilger on the normal hematocrit values as follows:

	%		%
Horse	34.2	Cat	40.8
Cattle	34.7	Swine	44.5
Sheep	32.5	Dog	50.4

Schalm and Wood,[4] give the range of healthy dogs as 35-55%.

Interpretation of Results

The hematocrit reading serves as an indicator of the number of erythrocytes and the blood hemoglobin value. From a series of 583 determinations on the blood of 540 dogs, Schalm and Wood[4] have compiled a

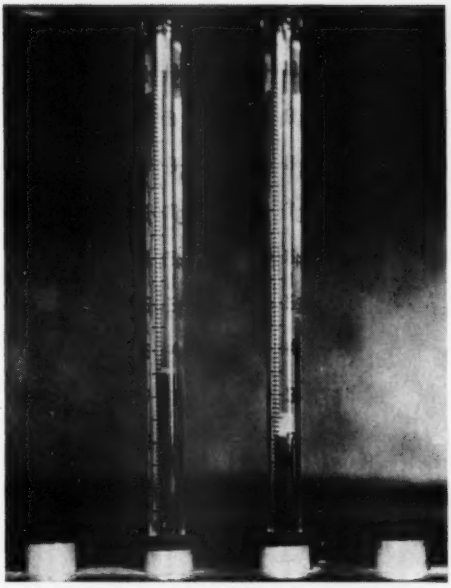


Fig. 3. Wintrobe tubes in position for sedimentation test.

table which predicts the total red blood cell count and hemoglobin value for any given hematocrit reading. The standard deviation is roughly 10% of the normal values.

The height of the column is influenced by the size and number of erythrocytes, as well as the state of dehydration of the animal. The hemoglobin content of the individual cells may also vary. With experience, the relative hemoglobin content can be estimated by viewing the column of red cells against a strong light. For these reasons, the hematocrit is only an approximation of the total red blood cell count and hemoglobin value.

Naturally, the hematocrit reading will be lower in cases of anemia. Such conditions as massive acute hemorrhage, chronic hemorrhage (severe hookworm infestation) and the hemolytic phase of leptospirosis will produce lowered hematocrit values. Schalm and Wood[4] state: "In general, the majority of canine anemias are related to a depression of the erythropoietic centers by toxic products emanating from infections, from nitrogen retention in chronic nephritis, or from displacement of the marrow by

pathological cells in leukemias." The hematocrit reading can not take the place of clinical observation as, in any of the conditions listed above, dehydration will elevate the reading and possibly be misleading.

Other valuable information to be derived simultaneously from this test, such as sedimentation rate, buffy coat layer, and icterus index, will be discussed in a future issue.

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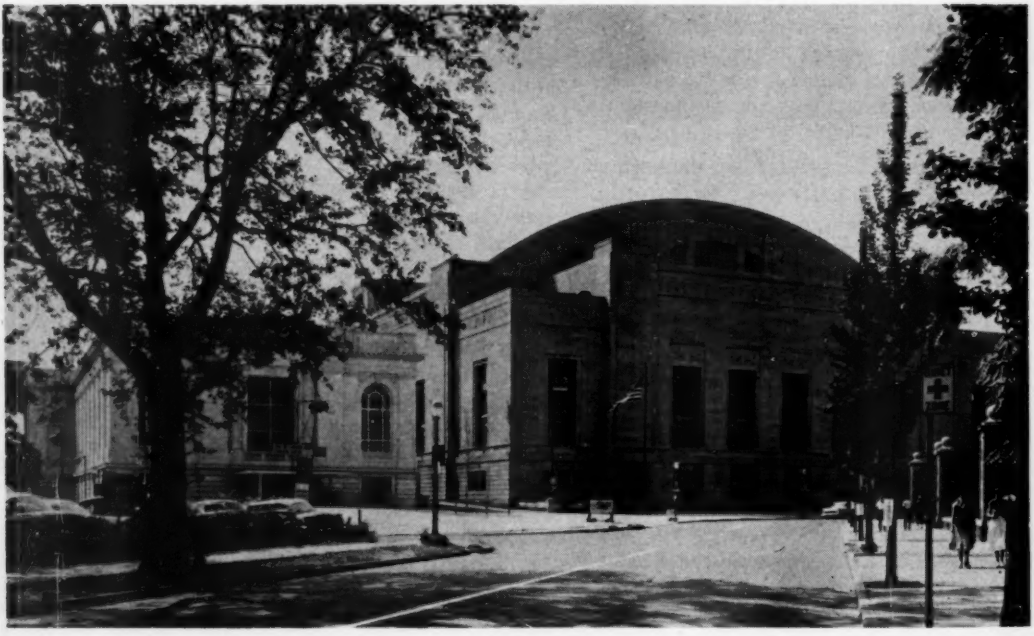
A.V.M.A. CONVENTION
AUGUST 18-21, 1958
Philadelphia, Pennsylvania

Pictured below is Penn Square Building, Philadelphia, center of activities for the 95th annual meeting of the A.V.M.A., Aug. 18-21, 1958. The Convention Hall, one of the finest in the nation, is within convenient access by surface transportation from all major hotels in downtown Philadelphia.

Announcement of progress of local arrangements and details for housing have been published. The Philadelphia Convention and Housing Bureau will handle all requests for reservations and those planning to attend are encouraged to make housing arrangements early.

Since the host city this year is conveniently located to several metropolitan areas and rural areas having higher than average concentration of veterinarians, convention attendence is expected to set a record.



Animal Health

R. R. DYKSTRA, D.V.M.

Cervical Abscesses in Swine

Gauge, *et al.* report on the effect of chlortetracycline on the incidence of cervical abscesses of swine in the *Journal of Animal Science* for February 1958. They indicate that out of approximately 50,000,000 swine slaughtered under federal inspection, 1,300,000 were condemned because of cervical abscesses including jowl abscesses, feeder boils, or strangles. They report that when the animals received a ration in which 50 gm. of chlortetracycline (Aureomycin®-American Cyanamide Co.) was added to each ton of feed, incidence of cervical abscesses was "significantly reduced." There also were weight gains. No better results followed the addition of 100 gm. of the antibiotic per ton of basal ration.

Radiation Hazards to Practitioners

Consultant in the Radiology Department of Medicine and Surgery, Ontario Veterinary College Doctor Broome is the author of an informative presentation entitled, How Dangerous are *X*-rays? (See *Canadian Jour. Comp. Med. Vet. Sci.*, Jan. 1958.)

It is indicated that younger operators are most likely to be harmfully affected since the action is cumulative. Over-radiation may lead to premature senescence, leukemia, osteogenic malignancy and/or radiodermatitis. All those using *x*-ray apparatus are potential candidates if precautions are not taken. The author indicates that the most hazardous machine in common use by veterinarians is "the small, light, self rectified unit of the dental type" as it requires short working distances, the spread of the beam is not limited by lead shutters, and the tube housing "may be much too light." Operators, and others should be as far as possible from radiation exposure, "wear a good quality leaded apron, leaded gloves, adaptation goggles, and use them always." Wear a monitor badge, and have it checked weekly by the National Research Council so that you may at once be made aware of overexposure — "the result is reported to you in milliroentgens." Do not expose a struggling patient as it may require too prolonged radiation and too frequent exposure. Animal patients should have the testicles protected by leaded sheets or pouches. Since the unborn young are always to be protected, except in an emergency, there must be no exposure of the mother as this invites congenital abnormalities. Doctor Broome expresses the hope that his article will not discourage practitioners from the use of *x*-rays as a diagnostic aid, but he also hopes that he has created perpetual respect for the wave length "you are using."

Hepatitis and Parasitism

Soulsby, of the Department of Pathology, University of Cambridge found no correlation between positive intradermal and precipitin tests and the presence of adult *Ascaris lumbricoides* in the intestinal tract in a test group of 273 pigs. This was confirmed by autopsy. However, there did appear to be a relation between positive reactions and the presence of chronic focal interstitial hepatitis. The writer of this column hazards the thought that there may be association of hepatitis with Ascaris infection.

Tranquilizers for Vicious Sows

Kristjansson of the Animal Husbandry Division, Experimental Farms Service, Department of Agriculture, Ottawa, Ontario Canada reports informatively on the Use of Chlorpromazine in the Treatment of Extreme Nervousness and Savageness in Farrowing Sows. (*Canadian Jour. Comp. Med. & Vet. Sci.*, Nov. 1957)

W. R. Hinshaw, D.V.M., Ph.D.

Poultry Practice

Poultry Feed Flavors

Dr. Moseley Kare of Cornell University in a lecture given at the 1957 Northeastern Poultry Producers Council (NEPPCO) meeting at Harrisburg, Pennsylvania in October, stated that although much research has been done on the tasting power and other senses of chickens, no one has found any "miracle flavors" favored by birds, (*The Poultryman*. October 1957). It is apparent that the discovery of preference for certain flavors would be of great value to compounders of feed and remedies in increasing acceptability of these items.

Doctor Kare reported that there is ample evidence that the chicken does have a sense of taste. The interpretation of flavors by the fowl appears, however, to be different than man. Some flavors that are disagreeable to man are readily acceptable by chickens, and in turn they may reject some that man considers agreeable. He emphasized that development of accurate methods for measuring response of animals to a flavor has been slow. Furthermore, even if feed can be made attractive to chickens by addition of flavors, there is still a question whether any advantage will come from catering to this sense. No broad spectrum flavor available which will improve taste of feed for chickens under all conditions has been found. In fact according to Doctor Kare, some commercial feed flavors tested by him were offensive to the chickens.

In liquid feeding, flavors are from 25 to 50 times more readily perceived by chickens. This fact is important when one considers the trend to "medicate" drinking water. It is particularly important that flavors objectionable to birds be determined in order to insure that normal consumption of water is not prevented by addition of drugs or supplements to the watering system. It is known that birds having access to normal non-medicated water as well as to certain medicated supplies will refuse the latter in favor of the normal supply.

Doctor Kare reported that chickens have an unusual ability to tolerate salt, but find it offensive in levels that might prove harmful, and will seek salt-free water if available.

Several years ago I found that turkeys consumed copper sulphate treated water in fairly high concentrations, but when fresh untreated water was available in the same pen, the birds refused water containing even slight traces of the chemical.

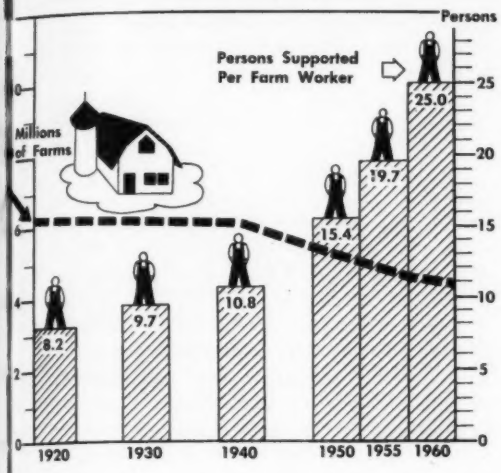
The above observations indicate that feeds or water to which drugs or feed supplements are added must be fully acceptable to the flock to be treated. In some cases, more harm than good may result if this precaution is not observed.

Chemicals obnoxious to birds might possibly be used in sprays to prevent cannibalism. Doctor Kare found that flavored sprays had no effect on preventing cannibalism, although some that were tried appeared to make picking by pen mates temporarily distasteful.

Poultry Management

Poultry Management in a Subtropical Semiarid Climate, USDA Production Research Report No. 5 issued in January 1957, should be of interest to veterinarians residing in southwestern United States. Burt W. Heywang, Animal and Poultry Husbandry Branch, A.R.S., in writing this manual draws from a lifetime of experience. He defines the area in U. S. as having such a climate as southern California, southern Arizona, and southwestern Texas.

CHART 1. Fewer Farms and Farm Workers Feed More People



The character of farm practice is being altered by the power of prevailing economic forces. Here the author analyses a previous report on economics (see VETERINARY MEDICINE, *53:173 [Apr.] 1958) and explains why instability of income, overproduction, and chronic poverty on some farms can result in the passing of both the free market and the independent professionalism of large animal practitioners.*

Farm Economics and the Veterinarian

BYRON L. JOHNSON, Ph.D.

Economics Department
University of Colorado
Boulder, Colorado

WHEN men see the character of society changing about them, they have two essentially different roads to take. They can describe and accept the change, perhaps even cooperate with it. Or they can detect in it certain dangers and call these to the attention of the public. And they can take steps to prevent the change from destroying basic values in the society they have known.

Veterinarians have already felt the impact of changes down on the farm. They have watched the growth of corporate farming, the development of contract feedlots, the decline of the truly independent family farm, and the attendant economic and social withering of the small rural community. They have seen the power of economic forces pour acid on the walls of their professionalism, and they have asked themselves an obvious question: Are these trends inevitable, or are choices still open to the profession?

Apparent Trends

In a report on Agricultural Trends and Veterinary Practice in the April 1958 issue of VETERINARY MEDICINE, economist Earl Crouse noted some apparent trends that are taking shape in the world of agribusiness. He pointed with interest to the development of supermarkets and contract farming as an efficient economic tool invoked to destroy the free market. He has forecast the consequences to the profession of veterinary medicine.

The Author . . .

Byron L. Johnson
Ph.D.

Dr. Byron L. Johnson is eminently qualified as an analyist in economic problems. He is Assistant to the Governor of Colorado and visiting lecturer in the Department of Economics, University of Colorado, Boulder. In addition to his report on agriculture, he has written numerous articles on taxation, state and local government, international trade, and other economic problems, in professional, trade, and popular magazines.

Doctor Johnson served as professor of economics, University of Denver, 1947-57. He has directly and indirectly served as an economist for various government agencies and has frequently presented testimony before congressional committees in Washington. He was statistician with the Wisconsin State Board of Health before serving in Washington. Doctor Johnson was also consultant to the New York Mayor's Committee on Management in 1951 and consultant to the City of Denver budget office in 1954.

In addition to his B.A., M.A. and Ph.D. from the University of Wisconsin, Doctor Johnson is holder of various honorary and scholastic awards. He is a member of the American Economic Association, the American Association of University Professors, and other professional groups.

Mr. Crouse based his article on certain premises that are evident; and at the same time he accepted some other premises, without stating them, that are not so evident.

The Farm Picture

Statistics have a way of becoming an obsession. The mass migration of one in 11 farmers to the city during a recent 12-month period must be understood in terms of certain facts, and the proper conclusions must flow from those facts. Low income farmers joining the ranks of factory workers in the city — or the ranks of the unemployed — do not mean that corporations are gobbling up the family farm at a rate fast enough to liquidate that type of unit in a decade or less. In fact, corporation farming is still a rarity, although the dangers inherent in developing trends can not be overemphasized. The farmer, as small business in agriculture, is suffering the fate of small business in the industrial world, and little is being done to check the trend.

Of the 134,000 farms with sales of $25,000. or over in 1954, a large share were individually owned. *"Commercial farming is still overwhelmingly a family business, and appears likely to remain so,"* or so stated the Committee for Economic Development in a recent report.

It is essential that we understand the farm picture as it exists in fact, not in political oratory. Census figures for 1954 revealed that there was a total of 4,783,018 farms in the United States. The implausability of developing for purposes of analysis a statistically average farm is noted with only cursory examination of table 1. Only 44% of all farms form the nucleus of the commercial farm class and constitute a significant factor in production. These market 91% of all farm products. The 56% that produce 9% can not be said to form a part of the economic picture, even though the social aspects of their problem have been a major concern of almost every farm program. Of the one in 11 that have left the farm during so short a period, almost all were owners or workers on submarginal farms.

Nevertheless these things are certainly true:

1. Productivity per man on the farm has been multiplied by bringing electricity and the wider use of power tools to the farm, just as it has been multiplied by use of better insecticides, custom designed fertilizers, and many other scientific and technological advances. (See Chart 1.)

2. As a result, the average size of farms and the investment per farm has climbed sharply, so that commercial farming requires much heavier capital investment than a generation ago.

3. The individual farmer does not exercise control over volume of production or current market supply, and is at the mercy of a market dominated either by supermarket chains or by the government, or else one subject to wide fluctuations.

4. In self-defense, particularly as farm income has steadily fallen, many farmers have accepted vertical integration — perhaps on the old theory that "if you can't lick 'em, join 'em."

To note these truths is not to endorse the development of vertically integrated livestock operation or crop farming. There was a time when most classically trained economists would have *come up fighting from their chairs* at the thought of monopoly growing unchallenged. Competition was accepted as worth while, and worth fighting to preserve.

Actually the invasion of this strange new phenomenon on the farm scene is merely an overflow of the success monopoly growth has enjoyed in other sectors of the economy. Examination of statistics accumulated during the past decade indicates that price increases have not necessarily been based on excessive demand for a variety of goods in short supply. The "administered price industry" has taken its place in the industrial as well as the farm economy. Such industries are powerful enough to shelter themselves from the type of competition normally associated with supply and demand, and can indeed write their own specifications, not only in quality, but volume and price as well.

Adjusting Factors

Examination of some other truths, not so widely understood or plainly stated these days, is also in order. They may point to why farmers and ranchers have abandoned their traditional freedom and independence for the apparent security of contract farming.

1. The aggregate demand for farm output is price inelastic. As output rises, prices fall faster. Hence a larger output will bring a smaller return than a smaller output.

2. The increasing output made possible by the new technology has faced declining markets despite a growth in population.

TABLE 1

	No. farms	% Total farms	% Total market sales	% Acres used
1. Commercial farms having market sales of:				
$25,000. or over	134,000	2.8	31.3	22.4
10,000. to 24,999.	448,945	9.4	26.9	20.8
5,000. to 9,999.	706,929	14.8	20.5	19.0
2,500. to 4,999.	811,965	17.0	12.1	14.1
TOTAL OVER $2,500.	2,101,839	44.0	90.8	76.3
2. Small full-time commercial farms having market sales of:				
$1,200. to 2,499.	763,348	16.0	5.7	8.8
less than 1,200.	462,427	9.7	1.4	3.9
TOTAL LESS THAN $2,500.	1,225,775	25.7	7.1	12.7
3. Part-time, residential and other* farms	1,455,404	30.4	2.0	11.0

SOURCE: Committee for Economic Development.
*This classification includes 2,693 institutional and experimental farms.

Farm Facts

There are many complicating factors involved in the evaluation of large animal practice outlook in the changing farm economy. Some of these are presented below. Others that run the gamut of farm statistics and social problems are too complex to be presented here. Almost all are essential, however, to the economist who tries to pinpoint the scope of trends and the complexion of change.

1. Farmers purchase some $17,000,000. worth of production goods annually, particularly machinery, fertilizers, feed, and similar products. This buying power can not decline year after year without hurting the economy.

2. Only 38% of farm income is bouyed by mandatory government price supports.

3. Analysis indicates that farm income is not a satisfactory index of whether the farmer can or will buy veterinary services. Census figures indicate that four out of ten farmers have income derived from activities off the farm — extra work, little enterprises, dividends, pensions.

4. Subsistence farming, even in low income farms, is not an easily recorded economic fact. Real income and money income frequently elude the best statistics. An arithmetic average of farm expenditures indicates that the average farm spends $8,400. annually. This is not, however, the real situation.

5. Rural "slums" are more of a social problem than a farm problem. Inclusion of these units in economic analysis of the farm situation without drawing attention to the proper relationship is frequently the cause for misleading results.

Contributing factors have been the end of war demand, curtailment of overseas shipments as a result of dollar shortages, improved production overseas, and trade wars.

3. Farming is subject to high fixed costs, but low variable or "out-of-pocket" costs — just the opposite of many commercial and industrial activities. Moreover, most of the fixed costs are implicit or "imputed." The farmer owes himself a wage. He owes himself his return on his investment. And he owes it to himself to build up depreciation reserves whenever possible. But if he can not meet these implicit costs — even if he consumes his capital — he will not be carried into bankruptcy. As a result, his minimum supply price is low.

This is the real reason why efforts to restrict output by lowering support prices are self-defeating. In the face of falling prices, each farmer is tempted to increase his output in order to maintain gross receipts to cover costs, and have a little left over. Under similar circumstances a manufacturer would shut down.

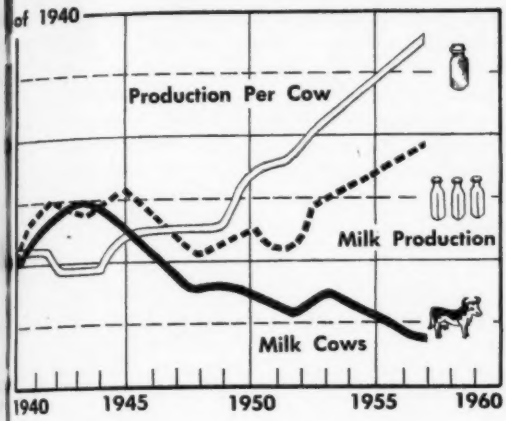
4. The farmer is thus the victim of his own efficiency. (See chart 2.) Every increase in productivity drives prices down and increases pressure on him to achieve still greater productivity to keep gross receipts up. Unlike a manufacturer of shoes, who can shut down or shorten his work week, the farmer and rancher have little choice — except under government control or under a private monopoly through producer cooperative control. The recent failure of the government to understand the farmers' economic situation is in fact leading to louder farm talk for producer monopoly controls.

The Future

There are two other truths that belong in the thinking of those who may be genuinely concerned about the future of American agriculture, and of rural society itself.

The first of these is that there are sound social reasons for preserving the family owner-operator farm system. The farm and its top soil are a heritage for future generations, a heritage better protected by an

CHART 2. Dairy Story in Visual Form



owner-operator who loves the soil than by a non-resident manager who loves only the profits he may be able to reap — even if reaping them involves mining the soil. Moreover, rural communities made up of owner-operators are stronger and healthier communities than those that depend upon migrant labor and the absentee landlord of a new corporate feudalism.

Should farm labor organize so that it no longer tolerates exploitation, part of the profit from corporate operation and vertical integration would disappear.

The history of vertically integrated enterprises demonstrates the penchant for taking profit at that level of operation that best serves its own interest. The supermarket chain can operate its feedlots to break the price of cattle, and destroy the independent farmer, at the same time making fat profits on the sale of meat over the counter. Federal antitrust prosecutions have amply documented this capability.

The producer who abandons his alternative outlets for the apparent advantage of a single buyer may be letting the camel's nose into the tent — and he may find himself forced out of the tent in the fulness of time. He is also helping the giant corporation gain complete control over the economy at the same time. *The passage of the free market is not an occasion for rejoicing, but for mourning.*

Consider the possibilities under monopoly. One real estate monopoly firm could handle all the transactions in any but the largest cities. One chain of supermarkets could sell all the groceries and meats needed. One dairy chain could handle all the milk, butter, and eggs a city, or the whole country requires. One dry goods store with branch outlets could sell all the clothing and household items needed.

One newspaper could bring "all the news that is fit to print." One radio-TV chain could furnish all the entertainment. One automobile manufacturer could produce all the models needed to supply the market. Shall we therefore encourage monopoly, and turn our backs on 68 years of antitrust legislation?

Recommendations

On the contrary, it seems to me that the American still really believes in competition, and wants to see it preserved. In agriculture, that means that these are the types of steps that must be taken:

1. Support those working to preserve the family farm, and support legislation that includes a family-sized farm unit cut off, so that government payments discourage rather than encourage corporate farming. This is something the veterinary practitioner can do as an influencial personage among farmer clients.

2. Discourage vertical integration by revision of the tax laws, so that farm losses can not be applied against corporate profits from non-farm activity. Question your representatives on this matter.

3. Support state legislation, as applied in Kansas, North Dakota, and Oklahoma, that seeks to break up or prevent corporate farming. Familiarize yourself with details of this legislation and discuss it with your clients.

4. Get antitrust enforcement of the Packers and Stockyards Act back in the hands of the antitrust division of the Department of Justice, where it will get disinterested enforcement — contrary to the practice of the Department of Agriculture, which now has jurisdiction. This is a legal matter.

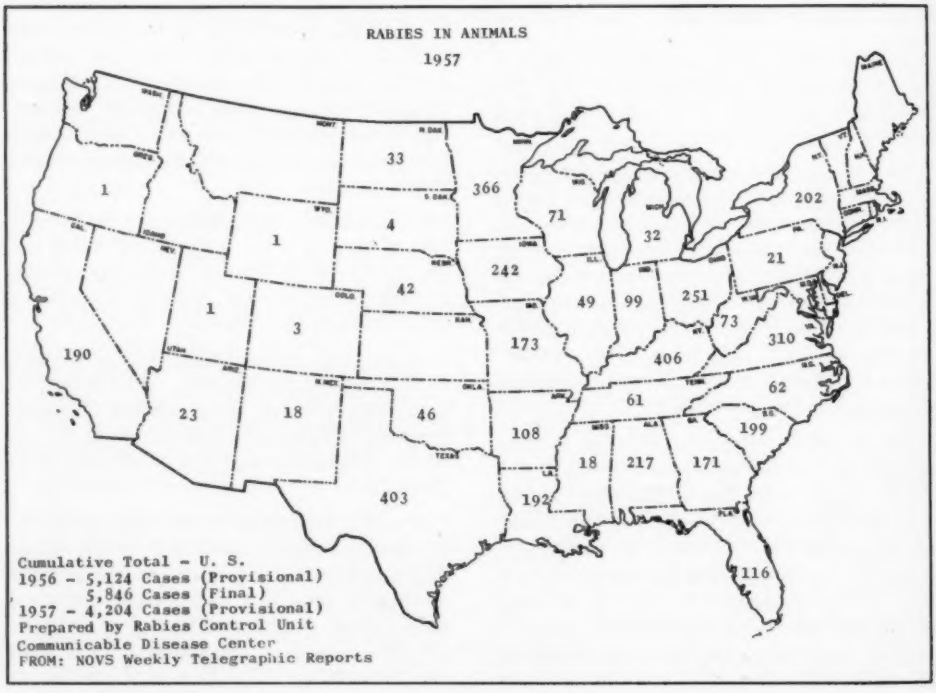
If, while these steps are taken, the farm policy of the government can be shifted from price support and protected domestic markets which restrict international trade over to policies favoring income support and freer trade with other nations — then the health of the American farm economy can be restored, and the farmer and rancher can continue its historically free and competitive way of life.

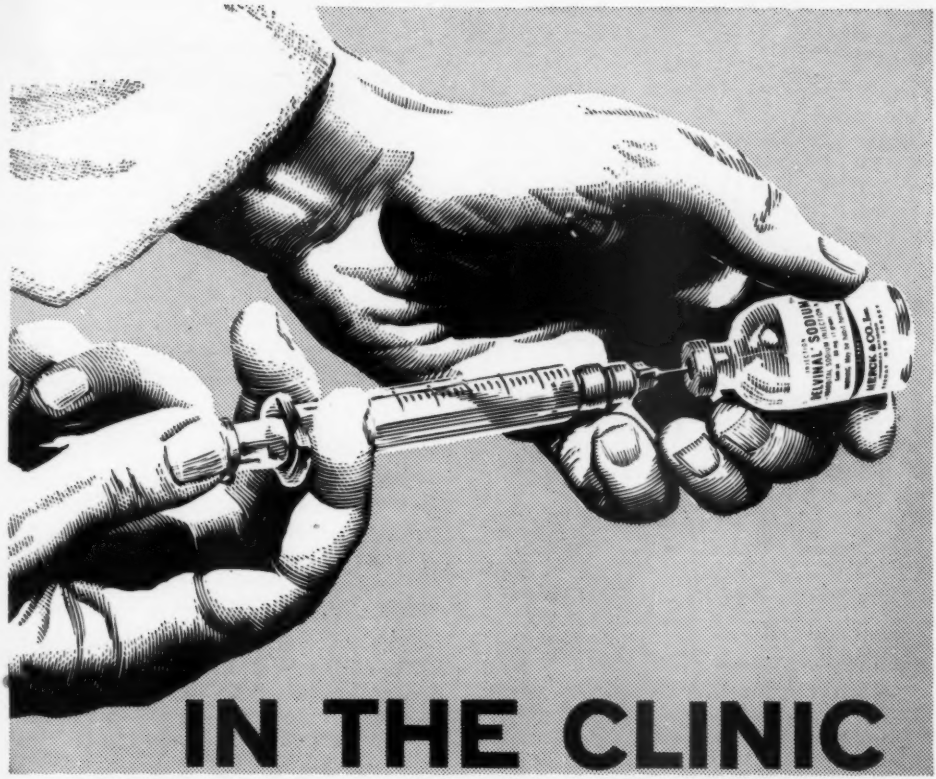
Veterinarians are not going to write the actual ticket — except as they are better informed citizens in daily contact with the livestock industry. Large animal practitioners can fall in with the trend toward integration, and expect in time to become hired hands themselves — or they can try to redirect the trend, and preserve their own independence as they preserve the freedom of the industry upon which they depend.

Elimination of Rabies

In its report to the public, the David Graham Hall Foundation of Texas states that outbreaks of canine rabies alert the public to temporary action; whereas sustained coordinated effort alone can be depended upon for realistic long-term rabies control and elimination.

Vaccination is not sufficient in itself — nor are quarantines — leash laws, and similar measures commonly proposed. The fact is, however, that vaccination is the basic fundamental which must be performed on every dog everywhere over three months of age, if indeed local outbreaks or rabies scares are to be forever eliminated. "There is an abundance of evidence," states the report, "that while epidemic years are usually followed by 'quiet' years, infected stray animals both wild and domestic, finally succeed in breaking barriers or crossing county or city boundaries, only to set off new chains of fearful apprehension among unsuspecting populations and disease among their unvaccinated animals whether under restraint or running free."





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·VM·

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Telephone: LEhigh 5-9188

Veterinary Examinations

Missouri

The Missouri State Board Examination has been scheduled for June 4-5 at the Veterinary Clinic, University of Missouri, Columbia. Veterinarians who wish to take the examination must contact Dr. L. A. Rosner, chairman, Missouri Veterinary Board, Box 630, Jefferson City, for an application.

Ohio

State Board examinations for Ohio will be given June 10-11 at the College of Veterinary Medicine, Ohio State University, Columbus. All applications must be made 30 days prior to examination date. License fee is $25. All applications should be addressed to Dr. H. G. Geyer, executive secretary, Ohio Veterinary Medical Board, Room 720, Ohio Departments Building, 65 S. Front Street, Columbus 15, Ohio.

Florida

The Florida State Board of Veterinary Examiners will hold an examination June 16-18, 1958 at the Biscayne Terrace Hotel, Miami. Inquiries should be addressed to Dr. E. L. Matthews, secretary of the Board, Box 141, Palatka, Florida.

Massachusetts

The Massachusetts Board of Registration in Veterinary Medicine will hold examinations for registration on June 26-28, 1958, at Amherst. The latest date for filing applications is June 20, 1958. All inquiries should be addressed to Dr. Edward A. Blake, secretary, Board of Registration in Veterinary Medicine, Room 33, State House, Boston.

South Dakota

The South Dakota State Board examination for license to practice veterinary medicine will be given June 23-24, 1958. The examination is scheduled to be given at the

South Dakota State College, Veterinary Science Department, Brookings, South Dakota. Applications must be in the hands of Dr. M. D. Mitchell, executive secretary, care of Livestock Sanitary Board, Pierre, no later than June 1.

Idaho

The Idaho State Veterinary Examining Board will conduct examinations for license to practice veterinary medicine and surgery in Idaho June 25-27, 1958. Applications must be received not later than May 25 at the Occupational License Bureau, State House, Boise, Idaho.

·VM·

If any of various additives currently employed in the packaging of food turn out to have long-range toxic effects, they may precipitate a large-scale disaster, according to Capt. Walter S. Diehl, U. S. Navy, Ret. —*S.N.L.*, 3/22/58.

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and THOMAS C. JONES, B.S., D.V.M.
Pathologist, Angel Memorial Animal Hospital, Boston; Formerly Chief, Veterinary Pathology Section, Armed Forces Institute of Pathology

GENERAL, special and systemic pathology of animal diseases is presented fully in this practical, authoritative work. It is believed to be the first concise treatise on all three phases of veterinary pathology. Basic essentials are followed by a discussion on diseases which have known causes or are due to injury or trauma, vitamin deficiencies, metabolic disorders, or poisons, especially plant poisons. Thirteen chapters are devoted to a consideration of diseases of body systems, with emphasis on those conditions in which the cause is in doubt. Recent advances in the field of veterinary medicine include the use of modern histochemical methods to identify specific pathogenic organisms in tissues and thus arrive at a precise diagnosis, how to recognize effects of atomic radiation upon animals and current data on how animals are affected by viruses.

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58-4

Dean Named at Alabama

Dr. James E. Greene, head of the Department of Clinics, Alabama Polytechnic Institute was named dean of the School of Veterinary Medicine effective March 1, 1958. Except for service with the Army Veterinary Corps (1942-1946), Dean Greene

Dr. James E. Greene

has been staff member of the School since 1937 when he served as graduate assistant in anatomy while pursuing his M. S. degree. He succeeds the late Dean R. S. Sugg.

In addition to his teaching duties Dean Greene has been active both in organized veterinary medicine and with various civic groups. His handling of many tasks have demonstrated his remarkable ability for leadership and he has given unstintingly of his time and talents. Beside numerous committee and board assignments, he served as president of the Alabama Veterinary Medical Association (1957). At present he is chairman of the Council on Education of the A.V.M.A. Dean Greene is exceptionally well qualified for the responsibilities of his new assignment.

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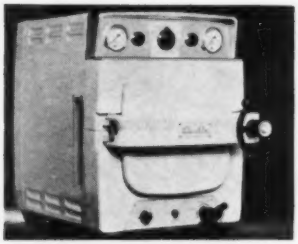

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filling, standby service, sterilizing, and venting are controlled by this single handle. Standby steam reserve makes it possible to use the unit as dry heat sterilizer for overnight processing of anhydrous materials. Weighing only 77 pounds and self-contained, it fits a 12¾ in. x 17½ in. counter or table top. For more information contact Wilmot Castle Co., 1901 East Henrietta Road, Rochester 3, N. Y.

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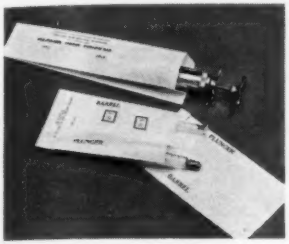

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·VM·

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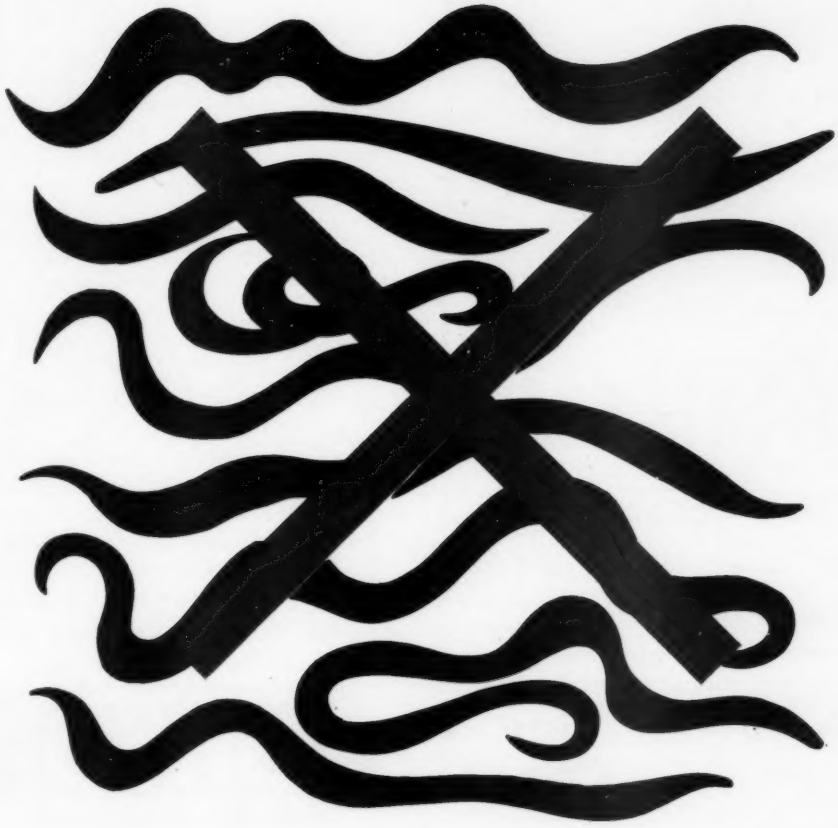

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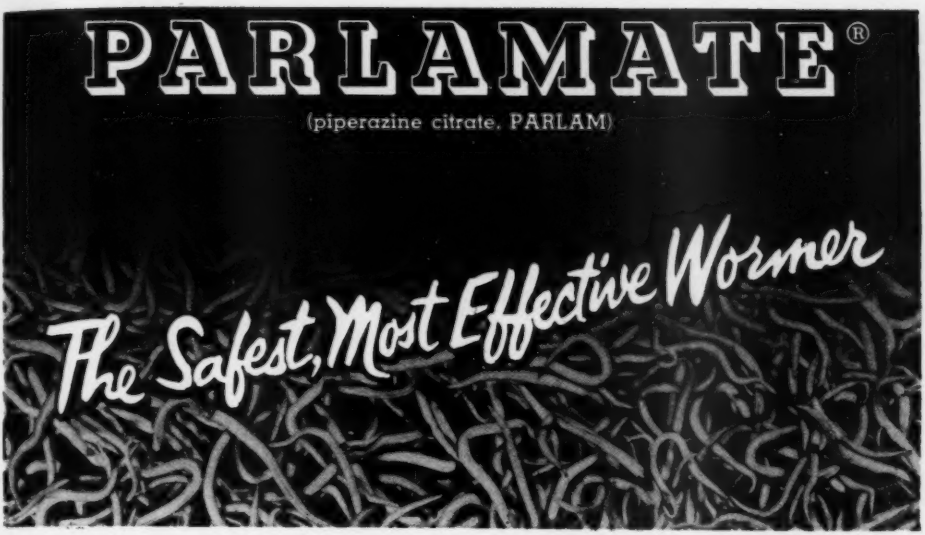

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Bibliography

1. Lynch, Donald J.: First Veterinary Symposium on the Uses of METICORTEN and METICORTELONE, New York, N. Y., Nov. 29-30, 1956.
2. Oberst, Fayne H., *ibid.*
3. Proctor, Delano L., Jr., *ibid.*
4. Chambers, Edward E., *ibid.*
5. Cutlip, Forrest W., and Kuhlmann, James H., *ibid.*

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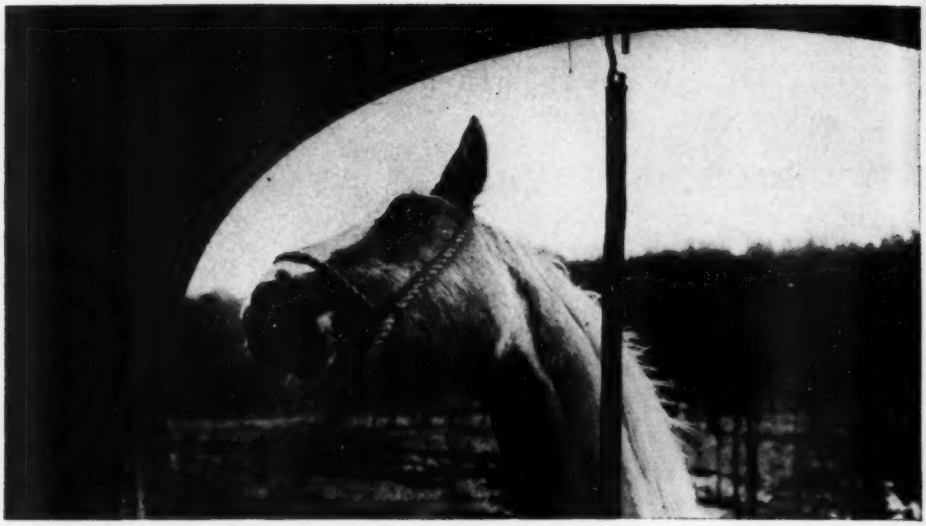

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Veterinary Calendar

May 5- 6. **Oklahoma Conference for Veterinarians.** College of Veterinary Medicine, Oklahoma State University, Stillwater, Okla. Dr. Lester Johnson, Department of Veterinary Medicine and Surgery, chairman.

May 6. **Eastern Iowa Veterinary Association,** 24th All-day Practitioners' Clinic. Hawkeye Downs, Cedar Rapids, Ia. Dr. F. E. Brutsman, Traer, Ia., secretary.

Jun. 2- 4. **Michigan Veterinary Medical Association,** Annual Meeting. Grand Hotel, Mackinaw Island. Dr. Charles Coy, Hillsdale, Mich., general chairman.

Jun. 5- 6. **Texas A. & M. College,** 11th Annual Texas Conference for Veterinarians. School of Veterinary Medicine, College Station, Tex. Dr. R. D. Turk, conference chairman.

Jun. 19-21. **Georgia and South Carolina Veterinary Medical Associations,** Joint Meeting. Bon Air Hotel, Augusta, Ga., Dr. A. Mills, 325 Pinecrest Drive, Athens. Ga., secretary.

Jun. 23-25. **California Veterinary Medical Association,** 70th Annual Meeting. Hotel St. Claire and Civic Auditorium, San Jose, Calif. Mr. Charles S. Travers, 3004 16th Street, San Francisco, Calif., executive secretary.

Jun. 23-25. **North Carolina State Veterinary Examining Board,** Washington Duke Hotel, Durham, N. C. Dr. James I. Cornwell, 65 Beverly Road, Asheville, N. C., secretary-treasurer.

Jun. 24-26. **Maritime Veterinary Associations,** Joint Conference. Mount Allison University, Sackville, N. B. Dr. J. F. Frank, Box 310, Sackville, N. B., general chairman.

Jun. 24-26. **North Carolina State Veterinary Medical Association,** 57th Annual Meeting. The Washington Duke, Durham, N. C. Dr. C. J. Lange, 3741 Hi-Point Road, Greensboro, N. C., secretary-treasurer.

Jul. 18-20. **Nebraska State Veterinary Medical Association.** Summer Meeting. Kearney, Nebraska. Dr. W. T. Spencer, 1250 North 37th, Lincoln, Nebraska.

Jul. 20-23. **Alabama Polytechnic Institute,** 51st Annual Conference for Veterinarians. Auburn, Alabama. (Housing facilities available for married couples.) Dean J. E. Greene, School of Veterinary Medicine, Auburn.

Jul. 21-23. **Canadian Veterinary Medical Association,** 10th Annual Convention. Royal Alexandra Hotel, Winnipeg, Manitoba. Dr. J. R. Singleton, 613 Dominion Public Building, Winnipeg 1, Manitoba, chairman.

Jul. 23-24. **Iowa State College,** Annual Conference for Veterinarians. Memorial Union, Iowa State College, Ames, Iowa. Dr. John B. Herrick, Ames, Iowa, extension veterinarian.

Aug. 10-12. **Mississippi State Veterinary Medical Association,** 52nd Annual Meeting. Buena Vista Hotel, Biloxi, Miss. Dr. J. W. Patterson, 221 Second Street, Columbia, Miss., in charge.

Aug. 18-21. **American Veterinary Medical Association,** 95th Annual Meeting. Municipal Auditorium. Philadelphia, Pa. Dr. J. G. Hardenbergh, 600 S. Michigan Ave., Chicago 5, Ill., executive secretary.

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—when diluted with water (except the tubercle bacillus)

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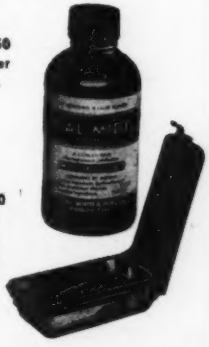

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Dr. Charles W. Turner, Missouri University dairy specialist and endocrinologist explains that thyroxin secretions vary fivefold from animal to animal. Because of this variation thyroprotein is effective in increasing milk production for those animals that do not secrete as much thyroxin as their bodies can utilize.

A cow at peak of milk production receives little if any stimulus from thyroprotein. However, through use of the synthetic hormone peak production can be maintained for a longer period with the result that total milk production per lactation is increased 20 to 30%. "It is most important," he concluded, "to give cows all the feed they will consume using thyroprotein."

•VM•

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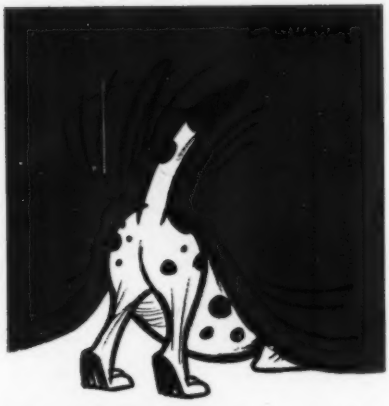

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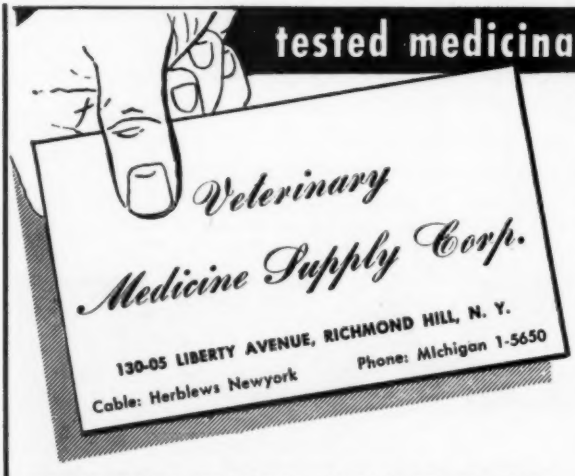

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Deaths of Veterinarians

G. W. KELMS, CVC '99; 35 years a practitioner in Milwaukee County, at Milwaukee, Wisconsin, July 19, 1957.

EWALD R. KLIMKE, WSC '53, 28; active in 4-H Club work; the Junior Chamber of Commerce and the Washington State and Puget Sound V.M.A., and was a member of the A.V.M.A.; of injuries received in an automobile accident, at Enumclaw, Washington, January 5, 1958.

A. W. KOESTER, 76; for 40 years in active practice; at Milan, Missouri, October 18, 1957.

CLARENCE A. KRAUSE, KCV '03, 75; with the Bureau of Animal Industry until 1938; more recently a practitioner at Portsmouth, Virginia; upon retirement in 1947 he moved to his home in Arizona; at Phoenix, January 19, 1958.

WALTER H. LEE, SF '13, 68; retired practitioner; former member of the A.V.M.A. and former president of the Wyoming V.M.A.; at Powell, Wyoming, December 18, 1957.

G. W. LITTLE, 64; practitioner; at Carnegie, Oklahoma, November 27, 1957.

ABRAHAM G. LIVENGOOD, GR '06, 76; the oldest practicing veterinarian in Pennsylvania; at Salisburg, Pennsylvania, November 11, 1957.

HAROLD S. MacDONALD, OVC '24, 57; prominent practitioner of Toronto; former vice-president of the A.V.M.A., member of the committee on diseases of small animals from 1944 to 1946; co-chairman of committee on reception and hospitality for the Toronto convention in 1953; member of the A.A.H.A., Canadian V.M.A. (president of Ontario Veterinary Association in 1943-44), and served as representative of his profession in the University of Toronto Senate from 1952-56; at Toronto, January 17, 1958.

HOWARD H. MALCOM, 56, practitioner; in an automobile accident at Ada, Oklahoma, November 11, 1957.

JOHN E. McCOY, KSC '09, 72, dean emeritus of the College of Veterinary Medicine, Washington State College, Pullman; private practitioner, veteran of the Army Veterinary Corps; member of the Washington State V.M.A. and the A.V.M.A.; at Walla Walla, Washington, January 15, 1958.

VELMA W. McDERMOTT, COL '48, Goleta, California.

BERNARD C. MEADE, NYS '16, 68; retired after service with USDA; at Stamford, New York, November 14, 1957.

O. RICHARD MEN'G. WSC '43, 38; member of Washington State V.M.A. and A.V.M.A.; at Four Lakes, Washington, December 5, 1957.

G. B. MERRICK, OSU '22, at Elmhurst, Illinois, recently.

WILLIAM R. MICHAEL, CVC '01, member of Illinois V.M.A., and A.V.M.A.; at Highland, Illinois, January 1952.

JONATHAN P. MILLER, STJ '20, 66, retired practitioner; past-president of the Missouri V.M.A. and member of A.V.M.A.; former Booneville City Councilman; at Booneville, Missouri, February 13, 1958.

EDGAR M. NEISWANDER, OSU '23, 63, general practitioner for 33 years; at Ada, Ohio, January 22, 1958.

BURIE E. PARKER, 62, practitioner in Mayfield for 30 years; at Mayfield, Kentucky, December 7, 1957.

VERNON S. PETERSON, ISC '42, 78; member A.V.M.A.; at Colman, South Dakota, December 17, 1957.

ROSS M. PHILLIPS, KCV '12, 78, poultry inspector for USDA; at Bertrand, Nebraska, July 18, 1957.

GEORGES H. RAJOTTE, MON '21, 58; at Montreal, Quebec, November 21, 1957.

KENNETH L. RITCHIE, ISC '33, 45; formerly with the United States ARS, USDA; member of the Iowa V.M.A., and the A.V.M.A.; at West Union, Iowa, December 1957.

GEORGE N. RUSSEL, KCV '11, 69; at Wichita, Kansas, December 22, 1957.

L. E. SHAY, STJ '09, 76; retired practitioner; at Atchison, Kansas, November 5, 1957.

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Deaths of Veterinarians

Continued

W. C. SCHOLT, ISC '02, 77; practitioner at Leon, Iowa; former state veterinarian; member of the Iowa V.M.A.; at Leon, Iowa, December 1957.

JOHN T. SCHWAB, STJ '23, 67; former state veterinarian of Wisconsin; past-president of the Wisconsin V.M.A., and member of the A.V.M.A.; at Tucson, Arizona, January 8, 1958.

P. B. SCOTT, CHI '06; at Galesburg, Illinois, November 10, 1957.

HARRY F. SEYMER, CVC '15, 66; at Milwaukee, Wisconsin, November 21, 1957.

FRANK SHICKLE, ONT '94, 87; active practitioner for 65 years prior to retirement a few years ago; at Bentonville, Arkansas, December 2, 1957.

FRED W. SHINN, ISC '14; 70; retired colonel; member of the A.V.M.A.; at Compton, California, December 3, 1957.

ABRAHAM N. SHIFRIN, OSU '12, 71; meat inspector for USDA from 1940 to 1955; at Cleveland Heights, Ohio, January 1, 1958.

D. L. SMATHERS, CVC '14; at Greenfield, Ohio recently.

ARTHUR E. SMEDLEY, CVC '06, 79; retired practitioner after 50 years; at Tallula, Illinois, January 26, 1958.

JOHN W. SMITH, KCV '09; at Lebo, Kansas, November 29, 1957.

WILLIAM G. STEVENSON, ONT '37, 44; of Guelph, Ontario, formerly with Ayerst, McKenna and Harrison, Ltd.; member of the firm of Stevenson, Turner, and Boyce Ltd., producers and distributors of ethical pharmaceutical products; member of AVMA, and on the committee on therapuetic agents; of a heart attack while traveling on business in the states, February 1, 1958.

J. ALEX STILL, 84; at Plant City, Alabama October 1957.

CHARLES D. TOFFLEMIRE, STJ '21, 68, former practitioner at Breckenridge and Cameron, Mo.; former inspector for Poultry Inspection service, USDA; at Marshall, Mo., October 28, 1957.

CORNELIUS VAN VLANDREN, ONT '03, Naugatuck, Conn., June 1957.

WILLIAM J. WALSH, KCV '18, 67, retired inspector of poultry for the USDA; member of A.V.M.A.; after a long illness at Creston, Iowa, September 27, 1957.

GEORGE D. WARREN, MCK '07, at Kankakee, Ill., April 17, 1957.

JULIUS G. WATTENBURG, KCV '13, practitioner and former meat inspector, at St. Louis, U. S. Bureau of Animal Industry; member A.V.M.A., Missouri V. M. A.; at Washington Mo., September 21, 1957.

HOWARD J. WATSON, KCV '17, at Sacramento, Calif.

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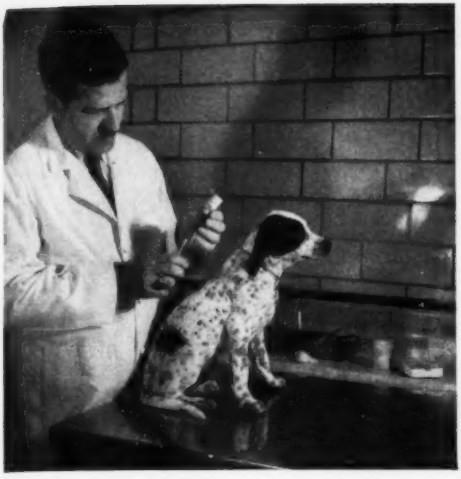

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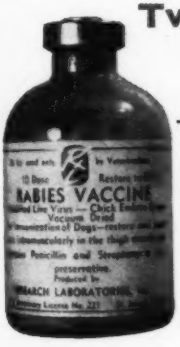

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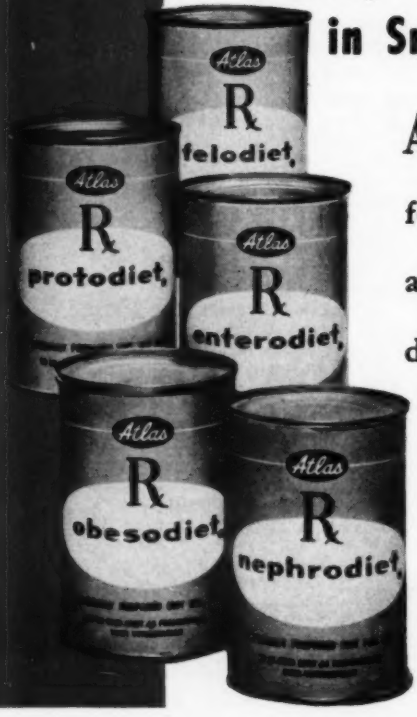

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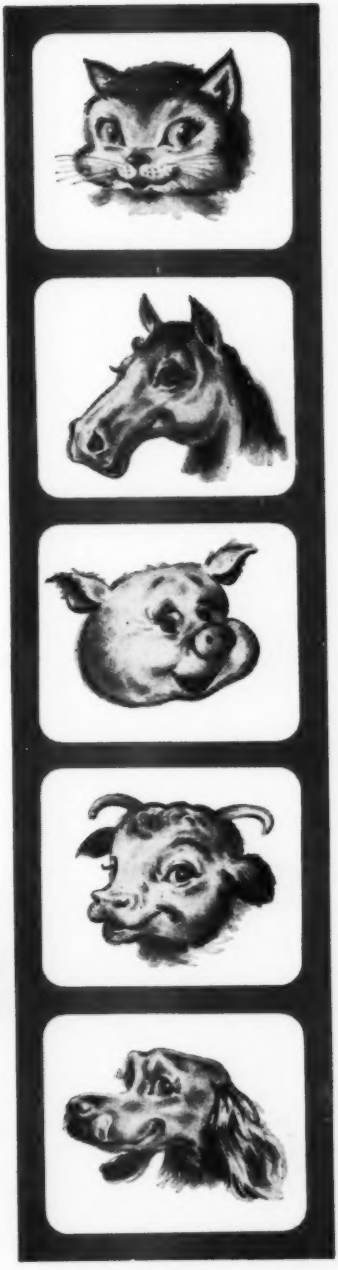

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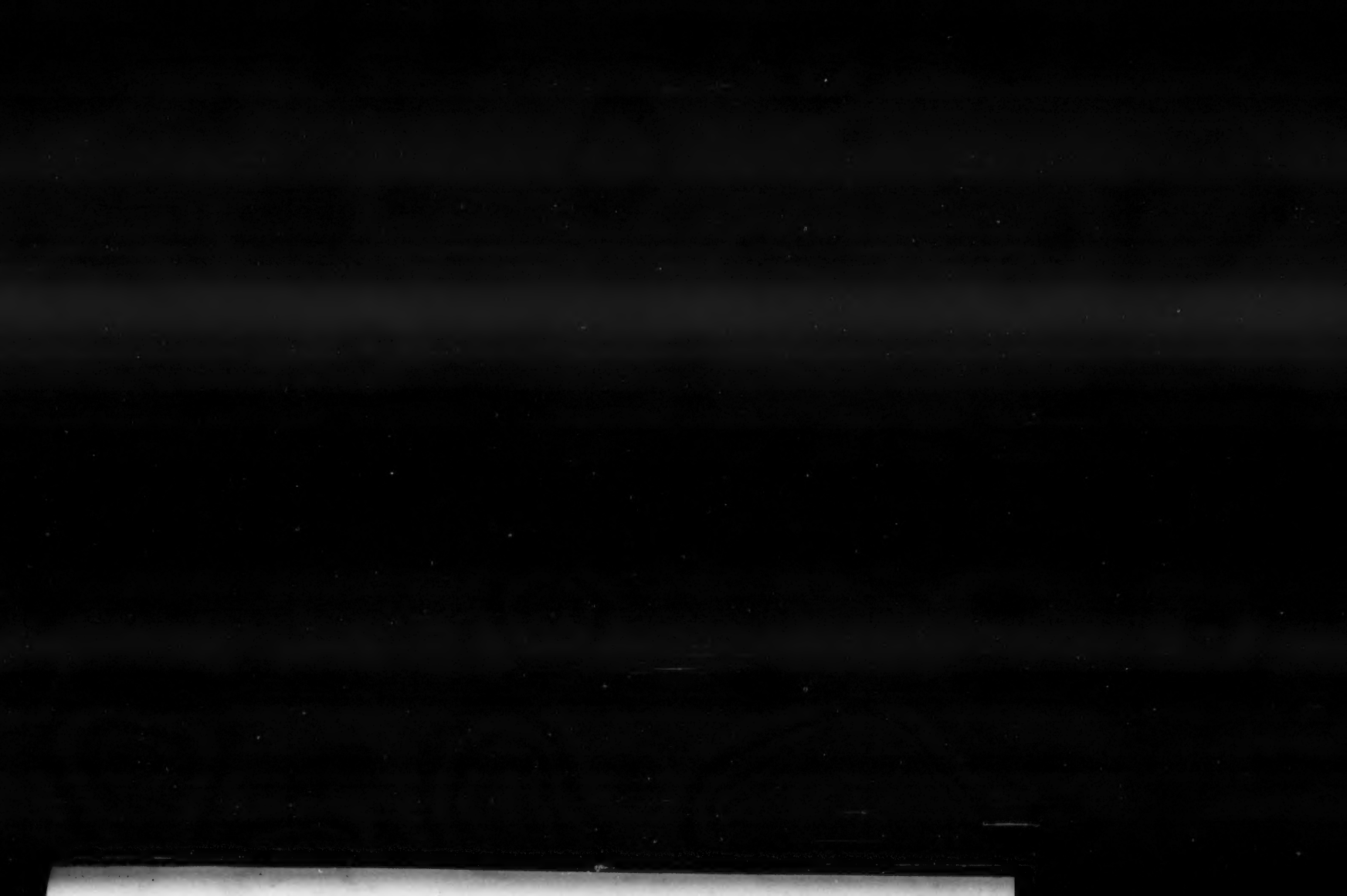
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